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HUMAN BIOLOGY

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MAY, 1930

THE INFLUENCE OF WEIGHT ON CERTAIN CAUSES OF DEATH

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I

It has long been known that the build of individuals has a direct bearing on longevity and, more especially, that persons extremely overweight or underweight suffer a mortality appreciably above the normal. Weight as a determining factor is clearly a component of two forces which, in practice, it is difficult to dissociate. The first is the congenital or hereditary element. This reflects the skeletal structure which the individual acquires from his antecedents. But once adult age is attained, body structure is, for the most part, fixed and, within narrow limits, determines the weight. The second factor reflects the peculiar dietary and living habits of the individual and is subject to control. This is superimposed, as it were, upon the congenital element and probably accounts for most of the variations in weight within the life span of the adult individual. In a country like ours, where life is easy and where food is abundant and relatively cheap, self-indulgence is very common and is probably the cause of much maladjustment and of illness. We are concerned then in this paper with an analysis of the effects of weight on longevity irrespective of the nature of the differences whether congenital or acquired.

* The writers' warm thanks are due to Dr. William Muhlberg, Medical Director of the Union Central Life Insurance Company, and Mr. E. E. Hardcastle, Actuary of the Company, for their kindness in placing at our disposal the records of their company for investigation.

While no proof is submitted in this paper, it is our opinion that by far the more important factor is that of inherited body structure.

It has been known in a general way that the degenerative diseases take their toll among overweight persons, and that the acute respiratory diseases and tuberculosis are the chief enemies of underweights; that, at younger ages, it is distinctly an advantage to weigh a little more than average weight, but that after thirty-five, persons of normal weight or less live longer than their adipose fellows.

Although these facts were more or less well known, particularly to insurance men, there has never been a systematic study of the causes of death according to build, based on a sufficient number of observations and with due regard to the important factor of age. A survey of medical and insurance literature during the past twenty-five years, shows but three important studies of the subject. The first of these was contained in the article "The Influence of Overweight and Underweight on Vitality," by the late Dr. Brandreth Symonds, the former Chief Medical Director of the Mutual Life Insurance Company of New York. But Dr. Symonds' survey suffered from a defect in method, the effect of which was to hide important differences which might exist between the weight classes in regard to the causes of death. A second study, similar in scope and method, was made by Dr. George R. Shepherd of the Connecticut Mutual Life Insurance Company. It was based on 26,000 deaths on which his company had paid claims in the period from 1846 to 1895.

The third study of this nature is part of that excellent and comprehensive mortality study—the Medico-Actuarial Investigation—a cooperative enterprise of the Association of Life Insurance Medical Directors of America and the Actuarial Society of America. The study covered the mortality experience of all the large life insurance companies of the United States during the years 1885 to 1909. In the published report of the study was a table showing crude mortality rates for several important causes of death by build in three broad groups of ages at issue of insurance. But this method, too, though better than that of the two earlier papers, was inadequate for the reason that it disregarded the effect of attained age, which is, after all, most important in evaluating death rates.

II

An adequate study of the diseases common to various types of build is needed. Fortunately we have had available a set of records

which gave us the material for such a study in ideal form. These were records of white men insured by the Union Central Life Insurance Company during the years 1887 to 1908.

This company is one of moderate size, operating largely in the Middle West, the South and the East. The men included in the study are, physically, better than average grade persons. They were all examined by physicians and found free from organic disease or serious impairments. Economically, they represent a relatively high standard, because the minimum insurance granted on their lives was $1,000. They are selected, too, in respect to occupation; all those in hazardous occupations being excluded. Thus these persons are not altogether typical of underweights, overweights and normals generally, but represent the best of each class.

Attention should be called to the period studied. It was a period of the most striking advances in public health conditions. Most of the improvement was in relation to infectious diseases of childhood, and consequently is not reflected in our study. Typhoid was the most important disease affecting adults which was conquered in the period, and, therefore, the high rates reported from it are only of historical interest today.

Another factor affecting our results is the reliability of the reporting of the causes of death during the period. Undoubtedly the reporting is better at the end of the period than it is at the beginning, because of the efforts of public health officers to improve certification of deaths. We are fairly certain on this account, therefore, that some causes of death are under-reported and others over-reported. Common instances are deaths from arteriosclerosis in which we now would get a record of heart disease; another, the reporting of terminal pneumonia where the true cause of death was some degenerative disease of old age. These inaccuracies do not affect our general conclusions, because they affect equally all weight classes, but it may affect the ranking of a few of the causes of death. It should be pointed out, however, that this insurance material is better than contemporary population data because the insured persons were of such grade that they usually would have a physician in attendance in final illness, and the diagnosis of the cause of death would be the best according to standards of certification then current.

The men for whom we have records numbered 192,304. Each of these individuals was traced from the time the insurance was issued on his life down to the policy anniversary in 1921 or the earlier termina-

tion of the insurance, for whatever cause. The experience in the first four years after issue of insurance was excluded in order to offset the effect of the medical selection of lives. Another result of this step was to limit the study to men twenty years of age and over. The 192,304 lives yielded down to 1921 an experience of 1,487,561 years of life or 7.7 years per individual. The experience of nearly one and one half-million life years was the same as an adult male population of that size observed for one year. There were 13,350 deaths in the group during the period of observation.

All these men were classified into nine weight classes according to the weight at examination for insurance. The basis of the classification was the normal or average weight figures of the Medico-Actuarial Investigation, already referred to. "Normal" or average weights were designated as Weight Class 0. Overweights were represented by Weight Classes I to V, Weight Class I representing those 5 to 14 percent in excess of normal; Class II, 15 to 24 percent; Class III, 25 to 34 percent; Class IV, 35 to 49 percent and Class V, 50 percent overweight or more. The underweights were represented in Weight Classes VI to VIII. Class VI represented 5 to 14 percent decrements from average weight, Class VII, 15 to 24 percent and Class VIII, 25 to 34 percent. Persons more than 35 percent underweight were excluded because the few in this class were found to be children.

The distribution of the cases and deaths by weight classes was as follows:

TABLE 1

Total Number of Men Insured and Number of Deaths Among the Survivors in the Fifth and Later Insurance Years Within Each Weight Class

WEIGHT CLASS	CASES	DEATHS
Total—all classes	192,304	13,350
Normal Weight		
0 (less than 5% above or below average)	64,260	4,131
Overweights (total)	38,062	3,876
I (5–14% above average)	25,932	2,353
II (15–24% above average)	9,494	1,137
III (25–34% above average)	2,323	332
IV (35–39% above average)	304	53
V (50% or more above average)	9	1
Underweights (total)	89,982	5,343
VI (5–14% below average)	71,947	4,264
VII (15–24% below average)	17,751	1,065
VIII (25–34% below average)	284	14

III

We have tabulated the experience of each weight class according to standard actuarial methods and we have calculated the death rates at the various attained age periods for each cause where the number of deaths was sufficient to warrant it. We have further calculated "standardized rates" which are derived by adjusting the death rates in each weight class according to a definite age standard, in order to eliminate the effect of differences in age composition between the various classes. The standard used was the age distribution of Weight Class 0.

At all ages combined and for all causes, the death rate for men of normal build in this experience was 844 per 100,000. For all underweight men, the death rate was 848 per 100,000; those most underweight (Weight Classes VII and VIII) had a mortality of 913 per 100,000, but those less underweight had a death rate of only 833 per 100,000 or slightly better than the normal group. The overweights, as a group, showed a mortality in excess of the normal group. Taking all of them together the death rate was 1,111 per 100,000. Divided into classes according to increasing weight, overweights showed the highest rates as the percentage of overweight increased. Weight Class I had a death rate of 1,027, Weight Class II, 1,215, and Weight Classes III–V combined, 1,472. Expressed as percentages of the death rate of average weight men, the mortality of those slightly underweight was 99 percent or approximately the same as the normal group, but that of extreme underweights, 108 percent, or 8 percent higher than the

TABLE 2

Deaths per 100,000—*All Causes—All Ages Combined By Weight Classes*

WEIGHT CLASS	DEATH RATES PER 100,000	PERCENTAGE OF RATE IN "NORMAL" WEIGHT CLASS
Underweights—Total (VI–VIII)	848	100
15 to 34% underweight (VII–VIII)	913	108
5 to 14% underweight (VI)	833	99
Normal weights (0)	844	100
Overweights—Total (I–V)	1,111	132
5 to 14% overweight (I)	1,027	122
15 to 24% overweight (II)	1,215	144
25% or more overweight (III–V)	1,472	174

normal. The mortality of overweights, on the other hand, ranged from 122 percent for Weight Class I to 174 percent for those excessively overweight. The penalty then for overweight is one-fourth to three-fourths excess in mortality. The facts are given in Table 2.

When the factor of age is included, the picture changes somewhat. At ages under 45, the penalty of overweight was not so great and the underweights did not show up to particular advantage. Men of normal weight showed the most favorable death rate, 463 per 100,000. This rate compares with 498 for all underweights, which is 8 percent in excess of normal, and 527 for overweights; or 14 percent above normal. It is noteworthy, however, that extreme underweights had a mortality at ages under 45 approximately the same as men of Build Class II, who are substantially overweight. Extreme overweights showed up badly, but the mortality of those only slightly overweight was but 10 percent in excess of normal and but 5 percent greater than among those slightly underweight. These results are shown in Table 3.

TABLE 3

Deaths per 100,000*—All Causes—Ages under* 45 *by Build Classes*

BUILD CLASS	DEATH RATES PER 100,000	PERCENTAGE OF RATE IN "NORMAL" WEIGHT CLASS
Underweights—Total (VI–VIII)	498	108
15 to 34% underweight (VII–VIII)	536	116
5 to 15% underweight (VI)	487	105
Normal weights (0)	463	100
Overweight—Total (I–V)	527	114
5 to 15% overweight (I)	510	110
15 to 25% overweight (II)	537	116
25% or more overweight (III–V)	682	147

It is at the older ages, after 45, that the death toll among overweights stands out clearest. The death rate in the normal weight class, was 1,308 per 100,000. Underweights as a whole showed a mortality of 1,274 per 100,000 or 3 percent better than normal. This favorable margin was due entirely, however, to the low mortality in Weight Class VI of 1,255 per 100,000, which is 4 percent better than normal. Extreme underweights had a mortality 5 percent higher than the normal group, and nearly 10 percent higher than those slightly underweight.

The mortality of overweights was far higher than any of these groups. Altogether, they show a mortality nearly 40 percent in excess of normals. Taken in increasing order of weight, the excess was 27 percent for Weight Class I, 56 percent for Weight Class II, and 86 percent for the extreme overweights who made up Weight Classes III, IV and V. The excessive mortality of these men is startling when it is remembered that they were most carefully selected when insured.

Table 4 gives the mortality of the groups at ages 45 and over, expressed as death rates per 100,000 and as percentages of normal.

TABLE 4

Deaths per 100,000—*All Causes—Ages* 45 *and Over By Build Classes*

BUILD CLASS	DEATH RATES PER 100,000	PERCENTAGE OF RATE IN "NORMAL" WEIGHT CLASS
Underweights—Total (VI–VIII)	1,274	97
15 to 34% underweight (VII–VIII)	1,372	105
5 to 15% underweight (VI)	1,255	96
Normal Weight (0)	1,308	100
Overweight—Total (I–V)	1,824	139
5 to 15% overweight (I)	1,658	127
15 to 25% overweight (II)	2,042	156
25% or more overweight (III–V)	2,436	186

The last three tables indicate very clearly the high mortality among those individuals whose weight shows a large departure from normal, particularly on the side of overweight. They show, too, the interesting change that takes place in the mortality of different weight groups as age increases. Thus, at the younger ages the mortality of overweight persons is relatively lower than it is later in life; among the underweights the opposite is true, namely, the higher mortality, as compared with other weight groups, is registered at the younger ages. In line with these observations is the fact that at ages under forty-five the most favorable mortality is found among persons of normal weight, but as age increases, the lowest mortality is observed among persons slightly underweight. Comparing the more extreme cases, we see that at the younger ages those showing marked underweight have a mortality corresponding to those 25 percent overweight, but at the older ages, these underweights do better than any group of overweights. It is very clear, from our tables, that extreme overweight is a distinct

hazard at every age period of life and this hazard increases with age. At the older ages, persons overweight even to a moderate degree have a mortality appreciably in excess of the average.

IV

Our main interest in this paper, however, is to point out the particular causes of death whose toll is significantly above or below the normal in the various weight classes and the extent of the departure in each case. We present here the facts on twenty important causes of death or groups of causes. Considering the overweights first, because their excess mortality is very large for all causes, we find that at all ages combined, they show exceptionally high death rates from organic heart disease and angina pectoris, arterial diseases, diabetes, nephritis and cerebral hemorrhage, and somewhat less excessive death rates from cancer, influenza, paralysis, appendicitis, cirrhosis of the liver and typhoid fever. With few exceptions, which will be noted later, these results hold true for both younger and older overweights. The favorable side of the mortality of overweights is the extremely low death rates from tuberculosis. The death rate from pneumonia, too, is appreciably lower among them than among lighter weight groups.

Conversely, when we turn to the underweights, we find that there is a huge excess mortality from tuberculosis, both of the lungs and of other organs of the body and also from pneumonia. Their mortality from all sorts of degenerative diseases, however, is quite low.

Persons of normal weight hold an intermediate position between overweights and underweights in their mortality from most causes of death. The only exception noted was the mortality from suicide, but even here, the difference from the underweight group was not very large.

The death rates from the most important causes of death among the overweights, underweights and normals are given in Table 5. It would not be amiss to bring to the reader's attention again that the differences between the weight groups are not differences due to age, because the rates shown in the tables have been standardized or adjusted to eliminate them. Therefore, the rates given are for groups which are identical in regard to age composition.

Let us next look into the extent of the excess mortality from each cause according to weight. We shall give first the diseases from which overweights show the highest mortality and then those that affect underweights most.

TABLE 5

Standardized Death Rates per 100,000 for Specified Causes of Death—All Ages Combined—By Weight Classes

CAUSES OF DEATH	DEATHRATE PER 100,000		
	Underweights	Normals	Overweights
All causes	848	844	1,111
Circulatory diseases			
Organic diseases of heart	65	80	121
Angina pectoris	14	16	35
Diseases of the arteries	17	23	38
Acute endocarditis and pericarditis	6	8	13
Nephritis, acute and chronic	63	82	141
Cerebral hemorrhage and apoplexy	49	70	110
Paralysis	12	14	17
Cancer	62	61	68
Diabetes	9	14	36
Tuberculosis, all forms	126	64	30
Pulmonary tuberculosis	115	57	26
Respiratory diseases			
Pneumonia, lobar and unspecified	70	63	59
Broncho-pneumonia	5	6	7
Influenza	20	20	28
Diseases of the digestive system			
Appendicitis	15	17	20
Cirrhosis of the liver	9	9	15
Typhoid fever	28	29	39
General paralysis of insane	12	11	14
External causes			
Accidents	55	60	67
Suicides	27	24	31

I. *Diseases of the Heart and Arteries*

Among normals and overweights diseases of the circulatory system caused more deaths than any other cause or group of causes. We have already pointed out that there is probably some understatement of heart disease as a cause of death and probably also of angina pectoris, but that, on the other hand, there is an over-statement of the number of deaths from arteriosclerosis. These facts should be borne in mind in the discussion.

At all ages combined, men of normal weight in the study had a death rate of 80 per 100,000 from organic diseases of the heart.

The death rate of underweights was 65 per 100,000 or only 80 percent of that for the normal group. For all overweights, the rate was 121 per 100,000, or 50 percent in excess of that for normals and nearly double the rate among underweights. Moreover, whereas the death rate from heart disease is quite stable in both groups of underweights, among overweights it goes up with increasing weight. The same situation was observed in the death rates from angina pectoris, pericarditis, and acute endocarditis. The death rate from angina pectoris increased from 14 per 100,000 among underweights and 16 among normals to 35 among overweights. The mortality of overweights from this form of heart disease, then, was more than twice the rate for the lighter weight groups.

The excessive mortality from organic heart disease among overweights occurred throughout adult life, although less marked at the younger ages. Under age forty-five, the death rate from organic heart disease was 18 per 100,000 for underweights, 25 for normals and 30 for overweights. The rate among the latter was thus one-fifth higher than among normals and two-thirds higher than underweights. At ages 45 and over, when the actual rates are much higher, the mortality among overweights from this cause was 58 percent in excess of normal and nearly twice the rate among underweights. Extreme overweights had in fact a death rate more than twice as high as that prevailing among underweights.

Similarly the death rate from angina pectoris among older overweights is twice that for normals and for the heavier weight men two and one-half times the normal. The mortality of underweights from this cause is nearly 20 percent better than that of the normal group.

Diseases of the arteries, too, showed a high death rate among overweights. Deaths from these diseases occur almost exclusively after middle life, and the few deaths at younger ages may be disregarded. At ages over forty-five, the death rate among all overweights was 78 per 100,000, compared with 45 among normals and 35 among underweights. The death rate for overweights was thus nearly 75 percent in excess of the normal and over twice that for underweights. The death rate among extreme overweights was nearly four times the mortality of the underweight group.

The facts regarding organic diseases of the heart are given in Table 6; for angina pectoris, in Table 7; and for arterial disease in Table 8.

TABLE 6

Organic Heart Diseases, Death Rates Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	65	81	18	72	123	84
VII–VIII	63	79	18	72	118	80
VI	66	83	18	72	123	84
Normal Weights						
0	80	100	25	100	147	100
Overweights—						
Total—I–V	121	151	30	120	232	158
I	115	144	28	112	220	150
II	135	169	37	148	254	173
III–V	129	161	*	*	255	173

* Not significant

TABLE 7

Angina Pectoris, Death Rates Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	14	88	2	67	27	82
VII–VIII	12	75	*	*	23	70
VI	14	88	2	67	28	85
Normal Weights						
0	16	100	3	100	33	100
Overweights—						
Total—I–V	35	219	7	233	69	209
I	32	200	7	233	62	188
II	39	244	*	*	79	239
III–V	37	231	..	...	82	248

* Not significant

TABLE 8

Diseases of the Arteries, Death Rate Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	17	74	2	40	35	78
VII–VIII	15	65	*	*	31	69
VI	17	74	2	40	36	80
Normal Weights						
0	23	100	5	100	45	100
Overweights—						
Total—I–V	38	165	5	100	78	173
I	34	148	*	*	70	156
II	46	200	*	*	97	216
III–V	41	178	*	*	75	167

* Not significant

II. *Nephritis, Acute and Chronic*

Breakdown of the renal system was the leading single cause of death reported among overweights and normals, but its outranking of organic heart disease is undoubtedly due to under-reporting of the latter. Moreover deaths from acute nephritis are included. The death rate from nephritis increased very perceptibly according to increasing weight. The rate for underweights of the group studied was 63 per 100,000, for normals 82, and for overweights 141. The last named, therefore, had a mortality 75 percent greater than men of average build and over twice as high as underweights. The lowest rate, 56 per 100,000, was recorded for the group 15 percent or more underweight, and the highest, 224 per 100,000 or four times as high, among men 25 percent or more overweight.

At ages under 45, the lowest death rate from nephritis was shown for the normal weight group. The rate for those moderately underweight is somewhat in excess of that for normals, but those extremely underweight had a mortality 40 percent in excess of the normal group. Overweights generally showed excessively high death rates from this

cause. Taken altogether, their death rate is nearly twice as high as normal but among extreme overweights nearly three and a half times as high.

At the older ages, underweight men showed particularly favorable death rates from this cause. The rate among the lightest weight men was only 56 percent of normal. Among overweights, on the other hand, the death rate was two-thirds higher than among the normal group. Those excessively overweight had a mortality two and one-half times that of normal and over four and one-half times that recorded for those 15 percent or more underweight.

The death rates from nephritis in each weight class and the relation of these death rates to normal are given in Table 9.

TABLE 9

Acute and Chronic Nephritis, Death Rates Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	63	77	26	118	108	69
VII–VIII	56	68	31	141	87	56
VI	64	78	25	114	111	71
Normal Weights						
0	82	100	22	100	156	100
Overweights—						
Total—I–V	141	172	43	195	259	166
I	108	132	33	150	198	127
II	202	246	70	318	363	233
III–V	224	273	75	341	406	260

III. *Cerebral Hemorrhage, Apoplexy and Paralysis*

Cerebral hemorrhage stood third among the causes of death of overweights. At all ages combined, the death rate in this class was 110 per 100,000, compared with 70 among normals, and 49 among underweights. Overweight mortality from this cause was thus 57 percent higher than among average weight men and over twice that of underweights. The mortality from this cause also increased

with weight; the lowest rate (46 per 100,000) was recorded for extreme underweights and the highest (170 per 100,000) for extreme overweights.

Although at the younger ages the death rate from cerebral hemorrhage was negligible, there was an appreciable excess mortality among overweights from this cause. At ages forty-five and over, it was a disease of major importance among normals and overweights, although among underweights it was only sixth among the causes of death. The death rate among them was 98 per 100,000; among normals, 134, and among overweights, 218. The death rate showed a constant change from weight group to weight group; for the lowest rate, 94 per 100,000 was found among those most underweight and the highest, 315, among those most overweight. In terms of percentage of the death rate for the normal weights, the increase was from 70 percent for extreme underweights to 235 percent for extreme overweights.

The death rates for paralysis run a course similar to cerebral hemorrhage throughout the life span, although the differences are much less marked.

TABLE 10

Cerebral Hemorrhage and Apoplexy, Death Rates Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	49	70	8	47	98	73
VII–VIII	46	66	*	*	94	70
VI	50	71	9	53	99	74
Normal Weights						
0	70	100	17	100	134	100
Overweights—						
Total—I–V	110	157	21	124	218	163
I	101	144	18	106	202	151
II	115	164	23	135	228	170
III–V	170	243	*	*	315	235

* Not significant

The death rates from cerebral hemorrhage and apoplexy in each weight class, together with the percentage comparison with normal weight, is given in Table 10.

IV. *Cancer*

Cancer was the fourth disease to show an excess mortality among overweights. In this case the excess was not so large as that observed in the degenerative diseases previously discussed, but was large enough to be significant and noteworthy. The highest death rates were recorded among overweights, but the death rate, at all ages combined, was approximately the same for both normals and underweights. Thus the rate for all underweights was 62 per 100,000, for normals 61, and for overweights 68 per 100,000; the mortality of overweights was slightly more than 10 percent in excess of the other groups.

At ages under forty-five, when the death rate from this disease was low, there was no appreciable difference between the groups. Thus the death rate among underweights was 21 per 100,000, among normals, 19, and among overweights, 18 per 100,000. There was

TABLE 11

Cancer (All Forms), Death Rates Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	62	102	21	111	111	99
VII–VIII	54	89	15	79	101	90
VI	64	105	23	121	113	101
Normal Weights						
0	61	100	19	100	112	100
Overweights—						
Total—I–V	68	111	18	95	128	114
I	64	105	16	84	121	108
II	73	120	20	105	139	124
III–V	86	141	*	*	146	130

* Not significant

thus actually a slight but not significant excess mortality among the lighter weight groups.

At the older adult ages, the picture was much clearer. Here the cancer death rate among overweights was appreciably higher than among the other groups, and it was persistently so. The most overweight group showed an excess of thirty percent above normals in the cancer death rate. The lowest rate was recorded for extreme underweights. There was, however, no marked difference between moderate underweights and normal weight persons in this respect.

The facts regarding cancer are given in Table 11.

V. *Diabetes*

Diabetes, more than any of the other important diseases, showed a great excess mortality among overweights. The result is not surprising because obesity, much of which is due to hypernutrition, is one of the most important factors in the etiology of diabetes. The death rates from this disease rose from 9 per 100,000 among underweights to 36 per 100,000 among overweights, but the seriousness of the situation among stout persons was displayed by the large increase in the death rate from 22 among moderate overweights to 117 among those excessively overweight. Expressed as percentages of the death rate of normals, overweights as a group have a mortality two and one-half times in excess of normal but extreme overweights have a mortality nearly eight and one-half times the normal, and thirteen times the rate shown for underweights. The underweight mortality from this disease was only 64 percent of the rate among normals.

Diabetes which is usually a disease with a gradual onset occurs mainly among older people. The death rate under forty-five is relatively low. The overweights in this young group have the highest death rate but it is not significantly higher than that shown among the other groups. At the older ages, however, the rate was much higher and it was here that great differences appeared. The death rate of normal weight persons at ages over forty-five was 23 per 100,000. Among overweights as a class, the rate was 69 per 100,000 or three times the normal. Among those excessively overweight, the mortality rate was 259 per 100,000 or over eleven times the rate for normals and over twenty times the rate observed among underweights. The death rate of the latter was only eleven per 100,000 or 48 percent of the rate among normal weight persons.

The facts regarding diabetes are given in Table 12.

TABLE 12

Diabetes, Death Rates Per 100,000 By Weight Classes and By Broad Age Groups

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	9	64	8	114	11	48
VII–VIII	9	64	6	86	*	*
VI	9	64	8	114	11	48
Normal weights						
0	14	100	7	100	23	100
Overweights—						
Total—I–V	36	257	9	129	69	300
I	22	157	8	114	40	174
II	45	321	*	*	84	365
III–V	117	836	..	...	259	1126

* Not significant

VI. *Cirrhosis of the Liver*

Cirrhosis of the liver, a relatively unimportant disease from the point of view of frequency, showed a high death rate among overweights. Among normal and underweight men, the rate was practically the same, 9 per 100,000, but the overweight classes, with a death rate of 15 per 100,000, showed a considerable excess mortality. The deaths from this cause were relatively infrequent at the younger ages, the highest death rates occurring after age forty-five. At these ages, the death rate among normals, 15 per 100,000, and among underweights, 17 per 100,000, were practically the same but overweights had a death rate of 27 per 100,000 or 80 percent in excess of normal. The death rate was even higher among those excessively overweight; that of Weight Class II (37 per 100,000) was nearly two and one-half times the normal.

VII. *Appendicitis*

Appendicitis, although very important from the point of view of morbidity, is relatively unimportant as regards mortality. The death

rates from this cause do not show much variation throughout the life span. The appendicitis death rate of overweights was moderately higher than that of the other weight groups. The excess mortality was largely confined to the younger ages. There was little difference in the death rates of normal weights and underweights.

VIII. *Typhoid Fever*

The findings regarding typhoid are today mostly of historical interest since the death rates are now so low. Our analysis shows, however, that, at all ages combined, the mortality of overweights from this cause was about a third higher than among men of normal build and that this excess was due to the exceptionally heavy mortality of younger overweights. At the older ages, the typhoid death rate was approximately the same in the three major weight groups.

. . . . .

Let us now turn to the diseases which showed excessive mortality among underweights. These diseases were primarily of pulmonary origin, with the exception of the relatively unimportant groups of deaths from extra-pulmonary tuberculosis.

IX. *Pulmonary Tuberculosis*

Tuberculosis is by far the most important disease affecting underweights. It far outranks every other disease among them, the mortality from it, at all ages combined, being nearly twice as high as that from heart disease, nephritis and pneumonia. Even at the older ages, when the tuberculosis death rate is supposed to be relatively low as compared with the organic conditions, it stands very near the head of the list of the major causes of death among underweights.

In our discussion, we shall concern ourselves with pulmonary tuberculosis only, which causes all but a relatively small amount of the total adult mortality from this disease. It might be noted here, however, that the mortality from other forms of the disease shows the same characteristics as pulmonary tuberculosis in regard to weight.

The death rate from pulmonary tuberculosis among all underweights was 115 per 100,000, compared with 57 per 100,000 among men of normal build and 26 per 100,000 among overweights. Underweights as a class thus had a mortality twice as high as that of persons of normal weight and four times that of overweights. There was, however, a distinct increase in the death rate from the disease with an

increasing degree of underweight. Thus, those in Weight Classes VII and VIII had a mortality of 166 per 100,000, and underweights in Weight Class VI of 103 per 100,000. The mortality among the lightest weight men was thus nearly three times the mortality of normal weights. The extent of the problem insofar as it concerns underweights, may be even better brought out by comparing it with the very favorable situation among overweights. In Weight Class I, the mortality was 28 per 100,000 or less than half that of normals and in Weight Class II, it was 21 or less than two-fifths the rate among normals, and among heavier overweights there were so few deaths in the experience that the actual rate was not significant. If we compare the death rates from pulmonary tuberculosis among underweights, with the rate of the middle overweight class, we find that the mortality in Weight Classes VII and VIII is eight times and in Weight Class VI five times as high as the rate among the overweights in Weight Class II.

The excess mortality of underweights from pulmonary tuberculosis is large throughout life. Compared with normals, the ratio of the death rates at younger and older ages is quite stable, but at the younger ages the death rate among all underweights was five and one-half times as high as that among all overweights. At the older ages the ratio of the death rates among underweights compared with overweights declines but it is still very large. Taking all underweights together, their death rate of 121 per 100,000 at ages 45 and over is nearly three and one-half times the death rate of 35 per 100,000 recorded for all overweights at these ages. The death rate in Weight Classes VII and VIII is five times the combined rate for all overweights and that of the underweights in Weight Class VI even is over three times as high as the overweight mortality at older ages.

A fact not usually realized is that, high as the tuberculosis death rates are among younger underweights, they are still higher among those over 45. Underweights as a class and normal weights also, suffered a mortality 10 percent higher at ages 45 and over than at the younger age. Among overweights the situation is even worse at the older ages, for the rate after 45 is 75 percent in excess of the rate before that age. Much of this increased mortality with advancing age was found among the higher weight groups.

The death rates from pulmonary tuberculosis in each weight class by broad age groups and the comparison with normal weight persons are given in Table 13.

TABLE 13

Pulmonary Tuberculosis, Death Rates Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	115	202	110	200	121	198
VII–VIII	166	291	158	287	176	289
VI	103	181	97	176	110	180
Normal Weights						
0	57	100	55	100	61	100
Overweights—						
Total—I–V	26	46	20	36	35	57
I	28	49	25	45	32	52
II	21	37	*	*	39	64
III–V	*	*	...	...	*	*

* Not significant

X. *Pneumonia (Lobar and Unspecified)*

Pneumonia likewise showed an excessively high mortality among underweights. We should keep in mind in discussing this disease that it is one of those which is possibly over-reported at the older ages, when it is only a terminal condition. This factor may explain part of the excess mortality from pneumonia found among underweights, some of which may really have been tuberculosis. In the same way we have no doubt that among extreme overweights many of the deaths reported from pneumonia were primarily due to degenerative diseases of the heart and kidneys.

The death rate among all underweights, at all ages combined, was 70 per 100,000 compared with 63 among normal weight men and 59 among overweights. The mortality of underweights from this cause is, therefore, 10 percent in excess of the normal group and over 15 percent in excess of overweights. The excess mortality of the extreme underweights is even greater. The death rate among them is nearly 50 percent in excess of normal. Men who are excessively overweight, however, also had a high death rate from pneumonia, although not as high as that found among the extreme underweights.

At the younger ages, the differences in the mortality from pneumonia among the three main weight groups were very small and not of any

significance. The differences noted above, therefore, are limited to the death rates among older persons. At ages 45 and over, underweights as a class had a mortality of 112 per 100,000, compared with 91 for men of normal weight and 87 for overweights. The mortality among underweights from this cause was, therefore, nearly 25 percent in excess of the normal group and 30 percent in excess of overweights. The extreme underweights had a mortality 69 percent in excess of that of normal weight men. At these older ages, the pneumonia death rate among moderate overweights is favorable; it is somewhat, though not significantly, lower than that of the normal weight group. Extreme overweights, however, had a mortality appreciably in excess of the other overweight groups and of men of normal weight.

The facts for lobar pneumonia and pneumonias of unspecified type are given in Table 14.

TABLE 14

Pneumonia (Lobar and Unspecified), Death Rates Per 100,000
By Weight Classes and By Broad Age Groups

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	70	111	35	88	112	123
VII–VIII	90	143	37	93	154	169
VI	66	105	34	85	104	114
Normal Weights						
0	63	100	40	100	91	100
Overweights—						
Total—I–V	59	94	36	90	87	96
I	56	89	33	83	84	92
II	64	102	50	125	81	89
III–V	72	114	*	*	128	141

* Not significant

The death rates from broncho-pneumonia, while very low, indicate a higher death rate among overweights than among men of normal weight or less. This, however, may be due to reporting of the disease when it was only the terminal condition.

XI. *Influenza*

The period of the study includes the influenza pandemic of 1918-1919 and the epidemic of the following year. It just misses the pan-

demic of 1889-1890. The effect of the more recent pandemic is not fully reflected in our experience, however, because there were few persons in it who were then under 45 years of age. The influence of this factor may be important and may explain the lack of definite characteristics in the mortality data according to age and weight.

At all ages combined we find an equally great excess mortality from influenza among extreme underweights and also among overweights as a class. The death rates in these groups were 29 and 28 respectively, or an excess of about 40 percent, compared with the death rate of 20 per 100,000 observed among normals. The death rate of moderate underweights from this cause, 18 per 100,000, was slightly but not significantly lower than that of normal weights.

The mortality of extreme underweights was high throughout the life span, approximately 40 percent above the normals both at ages under 45 and over 45. Men moderately underweight and those of normal weight had a relatively low mortality throughout life, the death rate in these two groups showing no great fluctuation by age.

Among overweights, however, the high rate recorded is limited to the younger ages, when it is nearly twice as high as the normal. At the older ages, on the other hand, overweights as a class have a

TABLE 15

Influenza, Death Rates Per 100,000

By Weight Classes and By Broad Age Groups

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	20	100	19	106	20	91
VII–VIII	29	145	26	144	32	145
VI	18	90	18	100	18	82
Normal Weights						
0	20	100	18	100	22	100
Overweights—						
Total—I–V	28	140	34	189	20	91
I	29	145	39	217	17	77
II	24	120	23	128	26	118
III–V	*	*	..	...	*	*

* Not significant

slightly lower death rate than normals. The relatively high death rate in Weight Class II is based on too few deaths to be significant.

As in pneumonia the high influenza mortality among extreme underweights may be due in part to deaths from tuberculosis, not reported, and among young overweights to deaths of persons impaired by bad hearts or kidneys or by diabetes.

The death rates from influenza in each age and weight class and the comparison with normal weights are contained in Table 15.

XII. *Accidents*

Death rates from external causes also showed differences worth observing. At all ages combined, the mortality from accidents was lowest among underweights and highest among overweights. It is realized that these differences do not represent biological variations but may only reflect the lack of alertness and agility of heavy persons. The death rate among underweights was 55 per 100,000, or only 92 percent of the rate for normals. Overweight mortality from this cause was 67 per 100,000 or 12 percent in excess of the normal weights and over 20 percent in excess of underweights. Furthermore there is a gradual but almost uninterrupted increase in the death rate from the lower to the higher weight classes. Thus the death rate in Weight

TABLE 16

Accidents, Death Rates Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	55	92	53	104	58	83
VII–VIII	44	73	35	69	54	77
VI	58	97	57	112	59	84
Normal Weights						
0	60	100	51	100	70	100
Overweights—						
Total—I–V	67	112	58	114	78	111
I	65	108	59	116	71	101
II	65	108	45	88	89	127
III–V	87	145	*	*	91	130

* Not significant

Classes VII and VIII combined was 44 per 100,000 compared with 65 in Classes I and II, and with 87 in Classes III-V combined.

At the younger ages, the death rates from accidental causes in the different weight classes show no marked variations, except for the very low rate among extreme underweights. After age 45, the picture is entirely different. Extreme underweights, as at the younger ages, and also those in Weight Class VI, had a low mortality. Overweights, however, were appreciably above normal. The excess mortality among them was limited to those more than 15 percent overweight. These older men in Weight Classes II-V had a mortality 30 percent in excess of normal.

The facts for accidents are given in Table 16.

XIII. *Suicides*

The death rates from suicide are much lower than most of the death rates we have previously discussed, and the absolute differences in the death rates between the weight classes are not very large. These differences are, to some extent, hardly more than we should expect in independent series of observations; but, on the other hand, certain of them are so characteristic that we feel that there is something significant in them.

The most favorable death rates from suicide were found among men who were of normal weight or slightly underweight. The mortality in these two classes was lowest both at ages under 45 and over 45. The mortality of extreme underweights and of all overweights was unfavorable throughout. At all ages combined the death rate in Weight Classes VII and VIII was over one-third in excess of normal. Among overweights as a class the mortality was also nearly one-third higher than among normal weight men. The high mortality from suicide of these extreme underweights and of all overweights characterizes the trend according to weight classes at both the younger and the older ages.

It may be that the excess mortality of these groups is due to the prevalence of illness. The suicide rate among disabled persons is high and it is not unlikely that the higher rate among underweights is connected with the incidence of tuberculosis and among overweights with that of the degenerative diseases.

The death rates from suicide in the various weight classes and the comparison of these death rates with the normal group are given in Table 17.

TABLE 17

Suicides, Death Rates Per 100,000 *By Weight Classes and By Broad Age Groups*

WEIGHT CLASS	ALL AGES		UNDER 45		45 AND OVER	
	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class	Death Rates Per 100,000	Percentage of Rate in "Normal" Weight Class
Underweights—						
Total—VI–VIII	27	113	21	124	34	103
VII–VIII	33	138	28	165	39	118
VI	25	104	19	112	33	100
Normal Weights						
0	24	100	17	100	33	100
Overweights—						
Total—I–V	31	129	25	147	39	118
I	31	129	24	141	40	121
II	29	121	26	153	32	97
III–V	42	175	*	*	48	145

* Not significant

V

SUMMARY AND CONCLUSIONS

In this paper, we have tried to establish the connection between build, as represented by weight, and the incidence of certain morbid conditions. It is clear that, in this respect, excessive weight is a great disadvantage, which increases with the degree of overweight and advancing age. The underlying cause of this high mortality among overweight persons may be in large part purely physical. The extra body bulk undoubtedly taxes the vital organs, putting a burden of work particularly on the heart, the kidneys and the blood vessels. The causal factors in arteriosclerosis, organic heart disease, nephritis and cerebral hemorrhage have much in common and often suggest the effect of body weight on the elasticity of the blood vessels. The theory of Virchow, modified by Aschoff, regarding the relationship between tissue fat and arteriosclerosis certainly fits the case of these overweights, at least in premature death from degenerative conditions. The high mortality of overweights may represent, too, structural malorganization, the extremes of which are seen in pituitary cases.

Overweights are apparently not well able to carry on bodily functions as efficiently as persons of lighter weight.

As we said at the beginning of our paper, overweight is largely a matter of hereditary constitution, and to that degree not controllable by the individual. Some of it, however, is often due to dietary habits and the mode of life. Overweight which results from these factors can, of course, be modified. In view of the high rates of morbidity and death among overweights, the necessity of continuous education of the public in these dangers is evident. The measures to control unnecessary overweight, through sensible diet and exercise, are usually simple and practicable. The emphasis should always be on habitual moderation, and not on the freakish diets which are followed for a time and then forgotten. Much harm has been done by the latter, particularly among young women, where ill-advised and over-much reduction has resulted in impaired health and vitality.

Underweight, biologically, is an asset. The underweight represents an economical type in respect to metabolism. The only serious disadvantage of underweight is the susceptibility to tuberculosis and the acute respiratory diseases. To some extent, the high death rates from these diseases among underweights may be limited to certain constitutional types. We know, for example, that very tall men suffer a mortality in excess of shorter men of proportionate weight. The high mortality from tuberculosis and pneumonia may be peculiar also to those types where underweight is pathological. Extreme cases are those found among persons with an over-active thyroid gland. These several types of low vitality are prey to tuberculosis and pneumonia, which it should be noted, are not caused by degeneration, but by the invasion of the body by harmful bacteria.

The present study, therefore, confirms the well known observation that persons who approach the mean of build and weight are best suited to withstand the hazards of life. In this country, the average weight is rather high, even among these insured persons. Consequently, the ideal norm is somewhat below the average used in popular weight tables for adults. Moreover, what we call the "normal" increase in weight with advancing age is not biologically normal at all and, therefore, should be guarded against. Longevity of itself is no great ideal, but it reflects usually a healthy and active life. The man is fortunate indeed who, despite an inherited disadvantage in build, lives out his normal life expectation; but doubly fortunate is he who, with the best type of physical inheritance, adds further to his years by adherence to the rules of health.

THE AGE AND SEX DISTRIBUTIONS OF PATIENTS AT THE MAYO CLINIC

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There are many questions in regard to the origin of certain diseases which might perhaps be solved if more could be learned about the incidence of these diseases in different countries, in different parts of one country, in the two sexes, and at different ages. Our attention was called particularly to the problem by the questions put to us two years ago by Dr. A. I. Ludlow of the Severance Hospital, Seoul. Having noticed that in Korea he was seeing few cases of cholelithiasis he next wanted to know whether this was due to a racial immunity or to some peculiarity in the way in which the patients coming to the hospital were being sampled from the nation as a whole. If an immunity could be demonstrated the next problem would be to find its cause, perhaps in dietetic customs, because if this were known, a means might be found of combating the tendency to the disease in Americans and Europeans.

As soon as Ludlow analyzed the age and sex composition of his hospital population he saw that the incidence of cholelithiasis should be smaller in Korea than in America, because in Korea sick old men and particularly sick old women are little inclined to consult foreign physicians and when they do they are generally too conservative to permit an abdominal exploration. Obviously, then, the morbidity from certain diseases in the Orient cannot be compared directly with that from the same diseases in America until a study has been made of the age and sex compositions of the groups seen by physicians in the two localities. Particularly when one is dealing with a disease such as cholecystitis, the incidence of which varies so markedly with sex and age, one must calculate what the percentage in the Orient would be if the hospital population there were constituted just as it

TABLE 1

Age and sex distributions of 25,343 new patients registering at The Mayo Clinic. The figures in the first twelve columns represent percentages of males and females counted in each month

AGE, YEARS	SEX	JAN.	FEB.	MAR.	APR.	MAY	JUNE	JULY	AUG.	SEPT.	OCT.	NOV.	DEC.	TOTAL CASES		PERCENTAGE OF TOTAL FOR YEAR		RATIO	PERCENTAGE OF SAME GROUP IN U. S.		TOTAL	PERCENTAGE	PERCENTAGE CUMULATED	YRS.
														MALE	FEMALE	MALE	FEMALE	M TO F	MALE	FEMALE	M AND F			
0–4	Males	2.40	2.01	2.36	2.50	2.61	2.80	2.45	2.22	2.36	3.14	2.47	2.84	326	.....	2.52	.....	1.09:1	25.4	.....	614	2.42	2.42	0–4
	Females	1.58	2.77	1.72	2.55	2.38	2.76	2.02	1.55	2.08	2.74	2.88	2.76	.....	288	.....	2.32	.....	.....	21.0				
5–9	Males	1.94	1.44	1.60	0.94	2.29	2.40	3.84	3.28	1.82	1.36	1.48	2.04	264	.....	2.04	.....	1.06:1	19.1	.....	502	1.98	4.40	5–9
	Females	2.37	1.49	1.72	1.44	1.95	1.74	2.41	1.94	2.60	2.16	1.54	1.50	.....	238	.....	1.92	.....	.....	17.6				
10–14	Males	1.57	0.76	1.98	1.77	1.54	1.90	2.56	4.13	2.25	1.36	1.18	1.69	239	.....	1.85	.....	0.90:1	18.5	.....	494	1.94	6.34	10–14
	Females	2.15	1.60	2.16	2.11	1.74	2.25	3.57	3.20	0.83	1.76	1.34	2.07	.....	255	.....	2.05	.....	.....	20.1				
15–19	Males	2.30	2.20	2.45	2.81	2.18	3.50	3.73	3.39	2.36	2.41	2.66	3.02	350	.....	2.70	.....	0.72:1	31.1	.....	817	3.27	9.61	15–19
	Females	4.07	4.16	3.56	5.33	2.90	3.78	3.76	4.26	3.54	3.94	4.63	3.11	.....	467	.....	3.76	.....	.....	41.0				
20–24	Males	6.64	5.46	7.26	5.52	5.86	7.50	6.18	5.40	7.08	4.82	6.41	6.30	801	.....	6.19	.....	0.79:1	73.8	.....	1777	7.01	16.62	20–24
	Females	8.25	8.10	6.25	7.66	8.50	8.80	7.52	9.40	7.81	5.00	7.83	8.75	.....	976	.....	7.87	.....	.....	85.5				
25–29	Males	7.93	10.35	10.09	7.92	8.79	8.30	6.82	8.58	8.37	8.38	8.49	9.33	1118	.....	8.64	.....	0.80:1	103.0	.....	2466	9.73	26.35	25–29
	Females	12.09	11.19	11.86	8.55	10.35	11.76	9.25	12.21	12.07	9.70	10.20	11.86	.....	1348	.....	10.86	.....	.....	134.8				
30–34	Males	12.91	13.03	10.37	10.52	11.57	9.40	8.42	11.33	11.27	12.04	10.76	10.66	1433	.....	11.08	.....	0.84:1	143.8	.....	3066	12.10	38.45	30–34
	Females	15.03	13.54	13.69	14.11	12.94	12.99	11.95	11.92	12.49	12.45	14.52	12.90	.....	1633	.....	13.16	.....	.....	173.0				
35–39	Males	13.09	13.99	10.47	12.50	10.93	12.11	12.15	10.69	9.87	11.93	12.04	13.32	1538	.....	11.87	.....	0.90:1	156.4	.....	3178	12.54	50.99	35–39
	Females	11.98	14.18	14.34	14.22	13.36	13.50	14.17	12.40	10.83	14.40	12.15	12.79	.....	1640	.....	13.22	.....	.....	186.4				
40–44	Males	12.35	11.69	10.47	12.29	12.31	11.61	10.90	11.33	10.41	12.35	14.01	11.90	1533	.....	11.85	.....	1.11:1	194.2	.....	2857	11.27	62.26	40–44
	Females	11.19	11.19	10.46	9.66	9.78	10.33	11.18	9.88	10.10	12.94	10.51	11.63	.....	1324	.....	10.67	.....	.....	181.0				
45–49	Males	10.69	10.06	9.90	10.63	10.82	8.50	9.27	9.95	10.31	8.17	10.06	11.63	1304	.....	10.07	.....	1.01:1	173.6	.....	2530	9.98	72.24	45–49
	Females	9.27	8.74	10.78	9.44	9.99	11.05	10.12	10.37	11.45	9.31	8.96	8.75	.....	1226	.....	9.88	.....	.....	193.2				
50–54	Males	8.85	9.67	9.99	10.00	7.99	9.40	9.77	8.89	9.33	10.57	9.08	8.88	1193	.....	9.26	.....	0.99:1	197.0	.....	2356	9.30	81.54	50–54
	Females	8.36	8.42	9.27	8.00	9.61	7.98	10.23	8.79	10.69	11.66	9.78	8.78	.....	[illegible]	.....	[illegible]	.....	.....	[illegible]				

50–54	Males	8.85	9.67	9.99	10.00	7.99	9.40	9.77	8.89	9.33	10.57	9.08	8.88	1193	.....	9.26	.....	0.99:1	197.0	.....	2356	9.30	81.54	50–54
	Females	8.36	8.42	9.27	8.00	9.61	7.98	10.22	8.72	10.62	11.96	9.89	9.33	.....	1158	.....	9.33	.....	.....	222.0				
55–59	Males	8.20	6.99	9.36	8.13	6.66	7.80	8.21	6.78	9.12	8.27	7.40	7.19	1003	.....	7.75	.....	1.15:1	221.4	.....	1843	7.27	88.81	55–59
	Females	6.21	6.18	6.68	7.33	6.86	5.22	6.84	6.88	7.81	6.57	7.93	6.57	.....	840	.....	6.77	.....	.....	211.2				
60–64	Males	5.62	5.75	6.51	6.88	8.69	6.40	8.31	6.56	6.76	8.48	6.51	5.59	806	.....	6.92	.....	1.60:1	238.4	.....	1432	5.65	94.46	60–64
	Females	3.73	5.01	4.63	4.78	4.80	3.89	3.47	3.88	4.06	4.90	4.33	3.92	.....	536	.....	4.32	.....	.....	159.6				
65–69	Males	3.50	3.93	4.62	4.79	4.37	5.20	4.05	4.87	4.81	3.77	4.54	3.28	556	.....	4.30	.....	1.60:1	214.8	.....	890	3.50	97.96	65–69
	Females	2.60	2.66	2.16	2.70	3.27	2.86	2.60	2.42	2.39	2.45	2.47	3.11	.....	334	.....	2.69	.....	.....	141.5				
70–74	Males	1.20	1.63	1.70	2.29	2.08	1.50	2.34	1.80	2.90	1.88	1.78	1.51	243	.....	1.88	.....	2.19:1	144.6	.....	350	1.38	99.34	70–74
	Females	1.13	0.64	0.75	1.11	1.16	0.72	0.48	0.68	1.25	1.08	0.62	0.46	.....	107	.....	0.86	.....	.....	66.0				
75–79	Males	0.74	1.05	0.85	0.52	0.85	1.30	0.96	0.42	0.54	0.94	0.69	0.62	103	.....	0.80	.....	3.64:1	100.0	.....	130	0.51	99.85	75–79
	Females	.....	.....	.....	0.55	0.26	0.31	0.19	0.29	0.10	0.49	0.10	0.23	.....	27	.....	0.22	.....	.....	27.5				
80–84	Males	0.09	.....	.....	.....	0.37	0.20	.....	0.21	0.32	0.11	0.39	0.09	21	.....	0.16	.....	1.34:1	53.2	.....	36	0.14	99.99	80–84
	Females	.....	0.11	.....	0.33	0.21	.....	0.29	.....	.....	0.10	0.10	0.23	.....	15	.....	0.12	.....	.....	30.0				
85–89	Males	.....	.....	.....	.....	.....	0.10	.....	0.11	0.11	.....	.....	0.09	4	.....	0.03	.....	.....	3.0	.....	4	0.01	100.00	85–89
	Females																							
90+	Males	.....	.....	.....	.....	0.05	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	1	.....	.....	90+
	Females																							
Total cases	Male	1085	1044	1060	960	1876	999	938	944	932	955	1013	1125	12931	.....	.....	.....	.....	.....	.....	25343			
	Female	885	938	928	900	1895	977	1037	1032	961	1020	971	868	.....	12412									
Ratio males to females.		1.23:1	1.11:1	1.14:1	1.07:1	0.99:1	1.02:1	0.91:1	0.92:1	0.97:1	0.94:1	1.04:1	1.30:1	1.04:1										

is in America. The arithmetic of such calculations is well known to statisticians, who use it every day in the comparison of death rates in various localities (Pearl).

While traveling about, visiting some of the leading medical centers in the United States, Ludlow was surprised to find that only here and there could he obtain the data which he needed to compare with his own. Seldom did he find a clinic registrar who could tell him the age and sex distributions of the patients being treated in his institution. Everywhere in the scientific journals he found reports of the sex and age incidence of various diseases but rarely was there a hint given as to the composition of the human material from which the samples had been taken.

It seems to us now that with more and more statistical studies being made everywhere, the need for surveys of patient populations must become more acute, and more and more investigators must see the necessity for beginning their work with basic analyses of their material.

SEX AND AGE DISTRIBUTION OF MAYO CLINIC PATIENTS

Table 1 and figure 1A show the sex and age distribution of 25,343 males and females, new patients who registered at The Mayo Clinic during the year from April 1, 1927 to March 31, 1928. The sample studied represents more than half of the new patients seen during this period. The ratio of males to females is 1.04:1. That the material is fairly homogeneous was shown by comparing the distributions of the first (approximately) 1,000 men and women registered each month with the distributions of the second thousand. There was little difference except that the "hump" in the women's curve at the age of fifty was more marked in the second group than in the first.

It will immediately be noticed that the health of women tends to fail from two to five years earlier than that of men. Because of this, practically all of the distribution area for the women is shifted slightly to the left of that for the men. The mode for the women is about the age of thirty-two years, while that for the men is about the age of thirty-six. More young women then young men are seen, and more old men than old women. There is a curious "hump" in the women's curve about the age of fifty years and another in the men's curve about the age of sixty. The possible significance of these humps will be considered later.

The percentage distributions shown in figure 1A represent a mixture of a local, small town and country practice and a widespread national and international practice. In order to separate the two groups and compare their composition we picked out the records of those persons

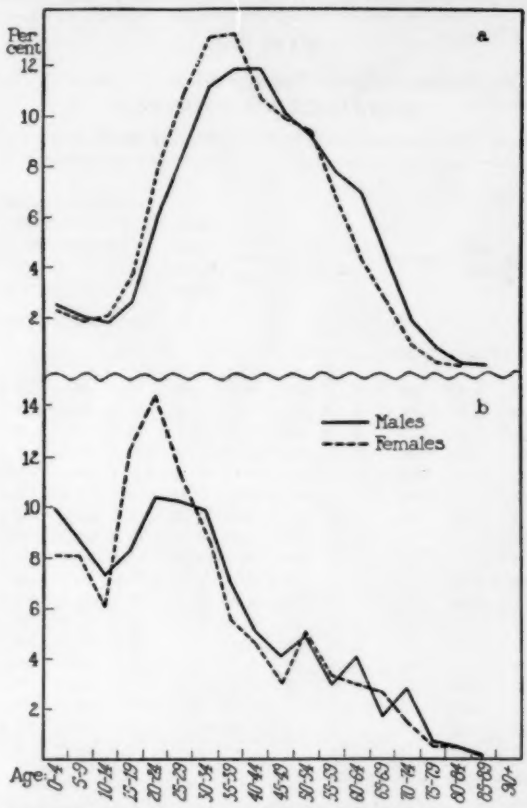

FIG. 1. *A.* POLYGONS REPRESENTING PERCENTAGE DISTRIBUTIONS ACCORDING TO AGE OF 25,343 MALE AND FEMALE NEW PATIENTS SEEN AT THE MAYO CLINIC DURING THE YEAR FROM APRIL 1, 1927 TO MARCH 31, 1928

B. POLYGONS REPRESENTING PERCENTAGE DISTRIBUTIONS ACCORDING TO AGE OF ALL THE NEW PATIENTS WHO IN THE YEAR FROM MAY 1, 1927 TO APRIL 30, 1928, CAME TO THE MAYO CLINIC FROM ROCHESTER AND FROM FARMS AND VILLAGES WITHIN A RADIUS OF THIRTY MILES

who during a year registered from Rochester and from the farms and villages within a radius of thirty miles. This distance was chosen because it represents about an hour's ride in an automobile, and it was thought that in these days of easy travel no one would consider

this much of a journey. In order to make the distribution curves more comparable with those representing the clinic practice as a whole we excluded records of children born, of women confined, and of nurses, physicians, and Clinic employees, who are examined when they go on the payroll and again at yearly intervals thereafter.

TABLE 2

Patients from Rochester and the Country within a Radius of Thirty Miles
May 1, 1927 *to April* 30, 1928

(*Nurses, physicians, Clinic employees, babies born, and mothers confined are excluded*)

AGES	MALES	PER-CENT	FEMALES	PER-CENT	TOTAL CASES	TOTAL PER-CENT	PER-CENT CUMU-LATED	RELATION TO MINNESOTA POPULATION PERCENTAGES			LOCAL CLIENTELE COMPARED WITH TOTAL CLIENTELE PERCENTAGES	
								Total	Male	Female	Males	Females
0–4	124	9.98	104	8.17	228	9.06	9.06	82.3	90.7	76.3	+7.46	+5.85
5–9	109	8.77	104	8.17	213	8.46	17.52	81.4	84.2	78.2	+6.73	+6.25
10–14	91	7.32	78	6.13	169	6.72	24.24	68.6	74.7	62.5	+5.47	+4.06
15–19	103	8.29	160	12.57	263	10.45	34.69	113.7	90.0	136.5	+5.59	+8.81
20–24	129	10.38	184	14.45	313	13.15	47.84	144.4	114.0	158.5	+4.19	+6.58
25–29	128	10.31	144	11.31	272	10.08	57.92	113.3	116.0	122.0	+1.67	+0.45
30–34	123	9.90	112	8.79	235	9.32	67.24	116.7	123.5	109.5	−1.18	−4.37
35–39	88	7.09	71	5.57	159	6.31	73.55	89.0	100.0	78.7	−4.78	−7.65
40–44	64	5.15	58	4.56	122	4.85	78.40	85.0	90.3	80.0	−6.70	−6.11
45–49	56	4.41	39	3.06	95	3.78	82.18	74.1	86.5	60.0	−5.66	−6.82
50–54	61	4.99	72	5.60	133	5.29	87.47	120.3	113.5	127.4	−4.27	−3.73
55–59	39	3.14	41	3.32	80	3.18	90.65	86.0	85.0	89.6	−4.61	−3.45
60–64	51	4.11	38	2.98	89	3.54	94.19	122.0	141.7	102.8	−2.81	−1.34
65–69	22	1.77	33	2.59	55	2.19	96.38	136.8	110.5	162.0	−3.53	−0.10
70–74	33	2.66	16	1.26	49	1.95	98.33	121.8	166.0	78.6	+0.78	+0.40
75–79	10	.81	9	.71	19	.75	99.08	125.0	135.0	118.0	+0.01	+0.49
80–84	8	.64	8	.63	16	.63	99.71	105.0	107.0	105.0	+0.48	+0.51
85–89	3	.24	2	.16	5	.06	99.77	30.0	120.0	80.0	+0.21	+0.16
Total.....	1242	99.96	1273	100.03	2515							

Ratio males to females 0.98: 1

Table 2 and figure 1B show that the part of the Clinic clientele which comes from the neighborhood and which represents a small town and country practice consists of patients considerably younger than those making up the national practice. This is shown also in figure 2A, which represents for the two sexes the cumulated percentage distributions according to age, and in figure 2B, which represents the differences between the corresponding fractions at different ages in the local clientele and in the national one.

The difference between the two clienteles would probably be even more striking if much of the house to house work in Rochester and the neighborhood were not attended to by independent general practitioners who call Clinic consultants only in case of need. In

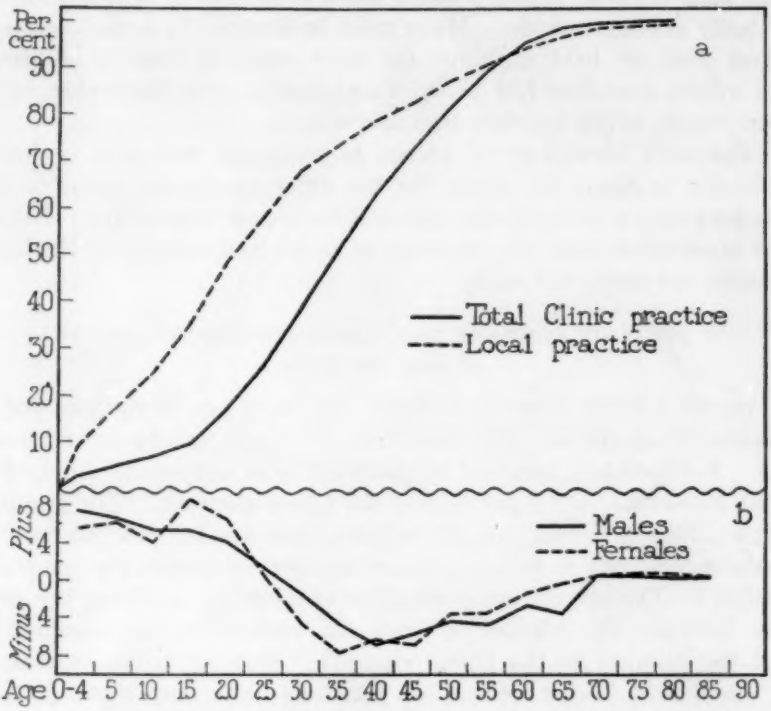

FIG. 2. *A.* SHOWS CUMULATED PERCENTAGES OF MALE AND FEMALE CLINIC PATIENTS WHO WERE DIVIDED INTO GROUPS ACCORDING TO AGE. The solid-line represents total clientele and the broken line represents patients from Rochester and the vicinity. The data used are the same as those presented in figure 1 but they are plotted differently so that one can see at a glance what percentage of the clientele falls below or above a certain age abscissa.

B. SHOWS FOR MALES AND FEMALES AT DIFFERENT AGES THE DIFFERENCE BETWEEN THE FRACTION IN THE LOCAL CLIENTELE AND THAT IN THE TOTAL ONE. For example, in the total practice there were 2.52 per cent of males aged less than five years; in the local clientele there were 9.98 per cent. The difference, +7.46 per cent, is the figure plotted.

view of this fact, it is the more noteworthy that a third of the community practice of the Clinic is made up of children and adolescents aged less than twenty years. A lesson that might be derived from this is that the general practitioner should be primarily a pediatrician

and that his teachers at college should see to it that he gets a particularly good training in this specialty.

Figure 1B shows that the modal age for both men and women is ten years lower in the local community practice than in the total one. This suggests that young persons when ill cannot or will not travel as easily as older ones do. Many must be deterred by a small income, others may be held at home by their responsibilities to children, and others doubtless fail to feel that concern over illness that, with good reason, is felt by older men and women.

The early breakdown of women as compared with men is shown strikingly in figure 1B where the big difference in the shape of the polygons comes between the ages of fifteen and twenty-five. Before and after these ages the numbers of males and females in the local clientele are about the same.

THE RELATION BETWEEN THE CLINIC POPULATION AND THAT OF THE COUNTRY

Figure 1 gives some idea of the age incidence of disease, but a moment's thought will show that this idea might well be an erroneous one. For instance, men and women aged from sixty-six to ninety-five years constitute only 5 per cent of the Clinic clientele. The group is small. Now is it small simply because there are but few old persons in the community or is it small also because old people are unusually healthy? The answer can perhaps be obtained by studying the relation between the number of such old persons in the community and the number in the Clinic clientele. Turning to the summary of the United States Census for 1920 one finds that white persons aged from sixty-six to ninety-five years constitute 4 per cent of the population. Obviously, then, this group supplies slightly more than its proper quota to the clientele of The Mayo Clinic.

In Figure 3B the lines represent the percentage distribution in 1920 of all white males and females in the United States. (Negroes are excluded because so few of them are seen at the Clinic.) In figure 3A each point in the two distribution polygons represents the ratio between the fraction of the Clinic clientele in that particular age group and the corresponding fraction of the males (females) resident in the United States. These new distributions look much like those in figure 1A. They show a tendency to be bimodal with, for women, peaks at the ages of thirty-seven and fifty-two years and, for men, peaks at the ages of forty-two and sixty-two years.

Figure 3C shows for the two sexes and the different age groups the ratio between the fractions of the clientele coming from the local community and the corresponding fractions in the population of

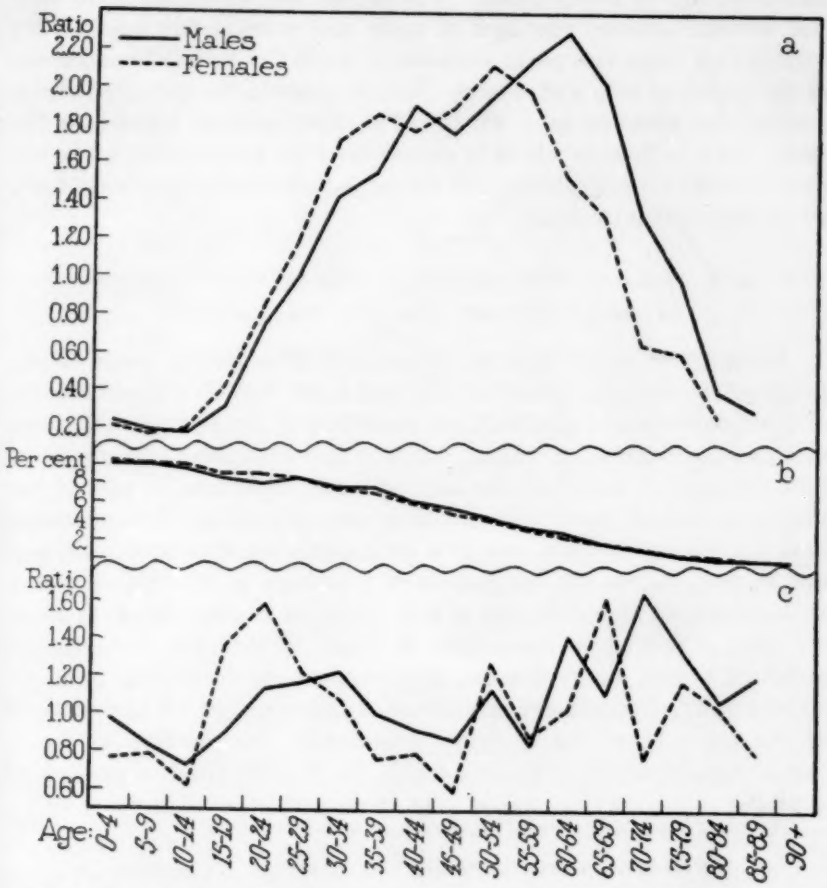

FIG. 3. *A.* THE LINES REPRESENT FOR MALES AND FEMALES AT DIFFERENT AGES THE RATIO BETWEEN THE FRACTION IN THE CLINIC CLIENTELE AND THAT IN THE WHITE POPULATION OF THE UNITED STATES IN 1920. For instance, in the Clinic clientele 2.52 per cent of the males were aged less than five years; in the United States 10.9 per cent of the population fell in this group. Two and fifty-two hundredths is to 10.9 as 0.25 is to 1.0 so 0.25 is the figure plotted.

B. REPRESENTS PERCENTAGE DISTRIBUTIONS ACCORDING TO SEX AND AGE OF ALL WHITE PERSONS IN THE UNITED STATES IN 1920

C. THE DATA PLOTTED WERE OBTAINED IN THE SAME WAY AS WERE THOSE SHOWN AT A BUT THEY SHOW THE RELATION BETWEEN THE SIZES OF THE VARIOUS AGE GROUPS COMING TO THE CLINIC FROM THE LOCAL COMMUNITY AND THE CORRESPONDING GROUPS IN THE POPULATION OF MINNESOTA

Minnesota (census of 1920). In these polygons the bimodality of the curves is more marked than in figure 3A. The first peak comes, for women, at about the age of twenty-two years, and, for men, at about the age of thirty years. The second one comes for both men and women between the ages of sixty and seventy-five years. We suspect that these two peaks more nearly mark the times of breakdown in the health of men and women than do those in the polygons representing the clientele as a whole. The first perhaps represents the early break in health which is experienced by those persons who are born with frail constitutions, and the second marks the final breakdown which terminates in death.

THE CAUSE OF THE IRREGULARITIES IN THE POLYGONS REPRESENTING SEX AND AGE DISTRIBUTIONS

In an effort to explain the appearance of a second mode in the polygons depicted in figures 1A, 3A, and 5, we took at random records of 1,668 patients and divided them according to the principal diagnosis into twenty-seven piles. Many diseases had of course to be combined into groups. A study of the distributions, according to age, of the men and women represented in these groups gave us the impression that the "hump" is due, not to a preponderance of persons with any one disease, but to the integration of a number of distributions each with a mode at about the age of fifty years for women and sixty years for men. The most important of these conditions were cancer, arthritis, goiter, heart disease, gastric ulcer, cholecystitis, prostatic hypertrophy, degenerative diseases of the nervous system and diseases of the eye. Since the complete solution of the problem evidently would require much time and study we decided for the present to drop it.

MONTHLY VARIATIONS IN THE CLINIC POPULATION

If in a private practice one were to study the records of 1,000 patients registered consecutively one would get an adequate cross-section of the clientele, but in an institution as large as The Mayo Clinic such a sample might easily give an erroneous idea because it would represent the type of persons who registered on only a certain few days out of the year. That a sample so taken would be inadequate can be seen from the fact that the age and sex composition of the patients coming to Rochester varies considerably from week to week.

In the northern states the arrival of winter brings with it a shut-down of active work about the farms; the men then have time to attend to chronic aches and pains, and as a result they come in large numbers

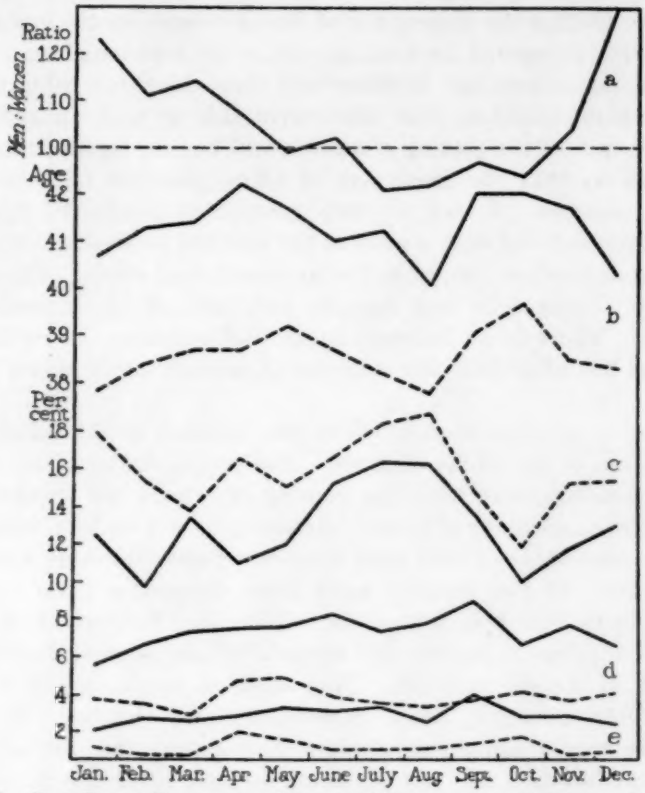

FIG. 4. THE LINES REPRESENT CHANGES NOTED FROM MONTH TO MONTH IN THE SEX AND AGE COMPOSITION OF THE TOTAL CLIENTELE OF THE MAYO CLINIC (APRIL, 1927 TO MARCH, 1928, INCLUSIVE)

A. Ratio of males to females.
B. Mean age. Solid lines represent males and broken lines, females.
C. Percentage of children and young adults of school and college age (from 5 to 24 years).
D. Percentage of persons aged sixty-five years and more.
E. Percentage of persons aged seventy years and more.

to the Clinic. In the spring and fall, when the men are working early and late, they have to put up with bearable discomforts and it is the women who come in. Figure 4A shows the way in which the ratio of males to females varies from month to month.

Besides the factor already mentioned there are several others that might affect the ratio between the number of men and women registered at the Clinic at different times of the year. One of the most important is the opening and closing of the schools. It will be noted in Figure 4A that the large influx of females comes on the first of July, shortly after closure of the schools. Many of these females are schoolteachers, but others are mothers and their daughters who, with the beginning of vacation, find themselves able to pick up and travel. The influence of the closing of schools will be seen again in figure 4B which shows that the mean age of Clinic patients falls during the summer months. It can be seen even more clearly in figure 4C, which represents for each month of the year the percentage of patients of school and college age, from five to twenty-four years. The number increases during July and August, and falls off in September and October. There is an increase again in November, December, and January, but after that the students apparently settle down to their work.

There is another way in which the weather might influence the composition of the Clinic clientele. Old people are so afraid of colds and pneumonia that with the coming of winter we should expect to see fewer and fewer of them. Actually, as will be seen from figure 4D, the percentage of men aged sixty-five years and more was lowest in January. It rose steadily until June; dropped a little, and then rose again to a peak in September. After that it dropped off for the winter. Strange to say the old women did not seem to fear the cold as much as the old men did. They came in largest numbers during the months of April, May, September, and October. For some unknown reason comparatively few came during the summer. Possibly grandmother was left at home to look after the house while mother and the children went to the Clinic. These peculiarities in the registration of old men and old women are shown again in figure 4E, which represents the percentage each month of patients aged seventy years and more.

Figure 5 shows twelve sets of polygons representing the age and sex distributions of approximately 2,000 new patients registered each month. Again it will be noted that more old men register in the summer months. It is curious that the second mode, which appears at about the age of sixty years in the men and at about fifty years in the women, is much more marked in some polygons than in others. In the case of the men it sometimes disappears

entirely, but in the case of the women it vanishes only in the polygon representing data from the month of January. Another curious feature is that with both the men and the women the location of this

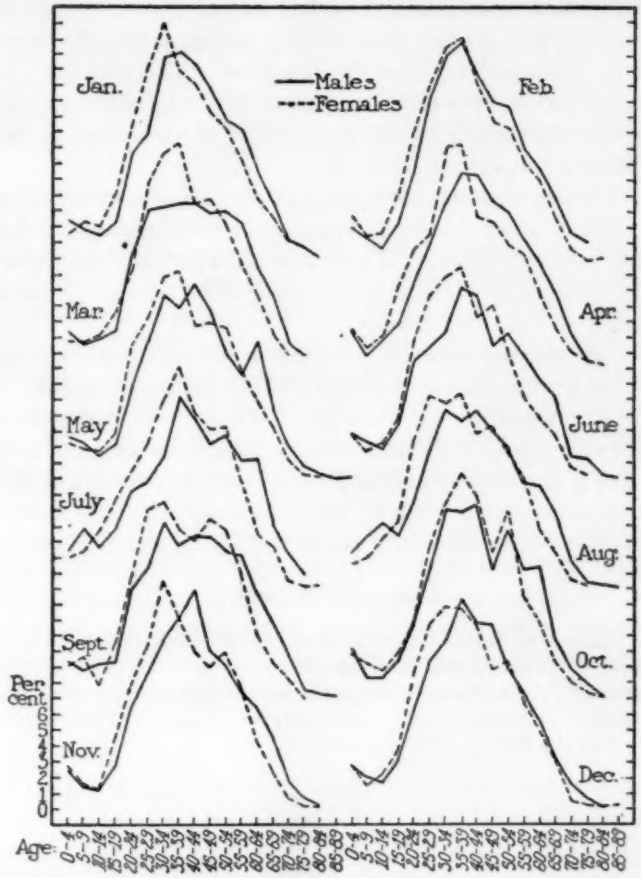

FIG. 5. PERCENTAGE DISTRIBUTIONS ACCORDING TO SEX AND AGE OF APPROXIMATELY 2,000 NEW PATIENTS IN EACH MONTH OF THE YEAR

"hump" shifts from month to month. These shifts show again that the exact location of the "hump" is largely a matter of accident.

SUMMARY

The incidence of various diseases in any two countries cannot safely be compared unless one knows the age and sex distributions of the clinic or hospital populations in the two countries.

Taking The Mayo Clinic clientele as a whole the ratio of males to females for the year, April 1, 1927 to March 31, 1928, was 1.04:1. The distribution polygons show a mode for the women at about the age of thirty-two years and one for the men at about the age of thirty-seven years. The whole distribution polygon for the women lies to the left (younger) of that for the men. There is another slight peak in the curves between the ages of fifty and sixty years, a peak which is due apparently to the summation at that time of large morbidities from a number of diseases.

The patients coming from Rochester and the country within a radius of thirty miles around average considerably younger than those who come from a distance. There is a large preponderance of women between the ages of fifteen and twenty-five years. Women break down earlier in life than men do.

There are marked monthly changes in the age and sex distribution of the patients, due to the weather and to the opening and closing of schools. A comparison of the size of the various age groups in the part of the clientele of the Clinic which comes from the local community and the size of the corresponding groups in the population of Minnesota shows that morbidity is high at two periods of life: one about the age of twenty-five years, the other about the age of seventy years.

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THE DECLINE IN THE AVERAGE LENGTH OF LIFE

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INTRODUCTION

In the issue of *Science* of July 26, 1929, appeared an article under the same title as that given above in which the writer referred to certain evidence that the average length of life may now be decreasing in this country. This was no more than a brief summary or report and included a promise that a longer article would soon follow. This promise is here fulfilled, but since the 1930 census, which is about to be taken, will soon enable us to replace many of the estimates used here, it would seem unwise to burden even this article with any part of the immense amount of routine work which has such a temporary value and—most of all—probably interests very few readers. This article then attempts to do no more than present the actual evidence of the previous article and clarify the processes and results. Although the numerical results are given here—in a somewhat isolated single table—no direct reference is made to them and the entire investigation is conducted by means of graphs of these results.

THE DATA

The data used in this investigation are the populations, by age groups, of ten states (the six New England states and Indiana, Michigan, New Jersey and New York), collected each ten years, and the corresponding mortality statistics, collected each year, by the Federal Government. These ten states are known as the ten original registration states and are the only states which have supplied the Federal Government with mortality statistics of requisite character every year since 1900. Other states have, of course, been admitted to the registration area since 1900 but, obviously, this investigation had to be restricted to the same area throughout. The period since 1900 is very short indeed, but it is the longest at our disposal unless we admit a few more data for 1890 which do not enjoy as good a reputa-

tion for accuracy as the later data and refer to only seven states—the ten enumerated above minus Indiana, Maine and Michigan.

PLAN OF INVESTIGATION

Most readers will probably be impressed by the absence of practically all direct reference to values of numerical results. It is only fair to devote some space to a short explanation of this attitude since it is deliberate. Stated concisely, we regard all of the Federal statistics used in this article—mortality and population—as very inaccurate—especially the distributions with respect to age—and we look upon all arguments over the identity of a digit beyond the first few as a waste of time. If this attitude is justified toward the original data, it is, of course, just as much so toward results deduced from them. This criticism of Federal data is most emphatically not directed against the Bureau of the Census. All of the credit for the data that we have belongs to the Bureau of the Census, and if the data furnished by most of the states are inaccurate or if, even, all of the forty-eight states are not yet enrolled in the registration area, by no stretch of the imagination can the blame be laid to the lack of effort on the part of the Bureau of Census. While such inaccuracies are to be deplored and should be remedied as soon as possible it is rankly unfair to expect a country so enormous and young to compete as yet with certain European countries which have been maintaining records literally for centuries. Compared with them we are still in swaddling clothes and they never did have the problem which we have.

Let us be frank and take a mere glance at the Federal mortality statistics. The essential requirement for a state or community to be admitted to the registration area is that its enumeration of deaths must be consistently "at least 90 per cent of all deaths"—that is, be correct to the nearest 10 per cent. The test consists in maintaining the records in two or more places and having the annual totals check within 10 per cent. Aside from the very unlikely possibility that all of the totals of a state may be badly off and still be acceptible because they check within 10 per cent, such a possible margin of error is obviously too large to justify any great refinement either in methods or results in any investigation based upon the data.

However, it is possible to be suspicious of refinements in results based upon Federal mortality and population statistics and still have considerable confidence in results in which reasonable allowances have been made for inaccuracies. In particular, such inaccuracies

should not be large enough to belie general *trends* in death rates or expectations of life from year to year. The general plan of investigation in this article then is to confine the attention, if possible, to the study and analysis of *graphs* of such trends.

After all, most of the graphs—particularly those of the males—are remarkably smooth considering the fact that no attempt whatever was made to smooth the original data in any case. In this connection, it is well to call attention to the fact that graphs of either death rates or expectations of life by ages are not to be expected to show the consistent trend year by year that they would by decennial years. Values for a specific year will, of course, be vitally affected by the occurrence of a serious epidemic—as of influenza—and these effects will show up strongly in a year to year comparison, whereas in a comparison by decennial years they become far less significant or noticeable. This distinction should be kept in mind in studying the two distinct parts of the interval covered by our data, considered in the succeeding pages.

THE METHOD

It is possible—although improbable—for one community to have a higher general death rate than another and yet have a longer average length of life. Such an apparent paradox would be due, of course, to differences in age distribution. The only way to investigate or compare mortality conditions of two or more communities, or—as here—of a community at different times, is to do so with distinct reference to all ages. Results so obtained constitute the essential items of a mortality table. This whole investigation is based upon the construction and comparison of a large number of abridged mortality tables.

Abridged mortality tables consist essentially of representative excerpts from the items usually associated with a mortality table. For extended applications in commercial work—as in life insurance—the whole mortality table and auxiliary information must, of course, be available, but for purposes of discussing the general health of a community, the items corresponding to several representative ages usually prove sufficient and since these items can be computed without constructing the whole table and in a very small fraction of the time necessary to construct the whole table, such a scheme proves extremely useful in an investigation like that undertaken here. The reader is referred elsewhere* for the derivation and explanation of the formulas

* Bulletin of the Amer. Math. Soc., Oct. 1929.

to be used in the construction of abridged mortality tables. The ages in the neighborhood of birth can not be included in abridged mortality tables. The ages in the neighborhood of birth can not be included in tables. The set of representative ages employed here are the decennial ages 10, 20, etc. It will suit our convenience considerably if we may be allowed to refer to a result which holds for these decennial ages as holding for all ages, although we shall be careful about taking this liberty unless there are strong reasons for believing the age of birth would not prove to be a serious exception.

Abridged mortality tables were constructed for males and females (separately) for each of the years 1900, 1910, 1920 for the ten states enumerated above, and for 1890 for the seven states specified above.

The mortality statistics were available for the successive years 1921, 1922, . . . 1928 but no population data were available since 1920. For these years the populations were extrapolated as follows: the well known formula

$$y = d + \frac{c}{e^{-ax} + b}$$

was fitted to the total (i.e., for all ages) populations for 1890, 1900, 1910 and 1920 for males and females (separately). Although there are certain restrictions which data must meet in order to be fitted by this formula no trouble was met in this investigation and the fittings were readily made since there are the same number of parameters as years. Then these totals were distributed into ten-year age groups (5–14, 15–24, etc.) in the same proportions which existed in 1920. That is, the proportion of the population for the age group 5–14 to the population at all ages, in 1920, was used to determine the population for the same age group for each of the years 1921, 1922, . . . 1928; and similarly for the other age groups.

It should be noted that the data and graphs of 1928 are additional to those referred to in the previous article in *Science*.

THE PERIOD UP TO AND INCLUDING 1920

It is unfortunate, but unavoidable, that we have such a short period of experience from which to deduce any conclusions as to how we are progressing in this country, in health or longevity. The period is so short that we feel compelled to use whatever data we can secure from 1890 even though the data for three states (Maine, Indiana and Michigan) are lacking and the data we have are not ideal. At that,

they are not greatly inferior, for our purpose, to those of later years. It is rather amusing to know that the writer will probably be criticised by some for the critical attitude he takes toward Federal data but that the most serious criticisms will be of results which he sifts from these data, which he must therefore in turn defend.

In the previous article the only reference to females was to the effect that the results "present the same picture—in somewhat less smooth form". This statement needs either proper interpretation or modification. The "picture" is the same but the development described later can not be said to have reached the same point as that of the males. When, however, we take into consideration the fact that females generally show a superiority over the males in longevity, there is evidence that the development to be described has reached the same point for females *relatively* as it has for the males. Nevertheless it is the males who present the clearest evidence and we shall give our main attention to them.

The following sets of graphs represent the expectations of life of each of three groups—males (of ten states), males (of 7 states) and females (of 10 states)—for each of five years: 1890, 1900, 1910, 1920 and 1926. The graphs for 1890, based upon only seven states, are included among those for ten states for whatever comparison the reader deems desirable. The explanation of the data and results corresponding to 1926, for each group, will be given in the next section. While, then, one is in no position just now to judge of the merit of these 1926 graphs, it is clearly an outstanding opportunity to introduce them when the paucity of graphs allows a vivid comparison —with a maximum of intervening interval.

Before satisfactory analyses of these graphs can be made something should be said about a simple device which is employed not only here but also in almost all of the other sets of graphs in this article. A serious difficulty encountered is similar to that of trying to plot heights of mountains and of houses using the same unit of measurement. Either the houses do not appear at all or the mountains extend beyond the limits of the graph. In our own immediate problem, the values at the earlier ages dwarf the values at the more advanced ages. Since our interest is avowedly in trends and not in the individual numerical values, it seems, and proves, preferable to plot differences between corresponding items, and it only remains to decide upon the most appropriate kind of base. Several kinds were tried, but the items for 1920 proved to be best—for several reasons—in all of the sets of graphs.

Thus, each item for 1920 was subtracted from the corresponding item—by age—of the other years and the differences plotted. In that connection, the reader should be prepared, at the start, for the generally accepted claim (considered later) that 1920 was probably an abnormally "good" year—on the grounds that it probably reflected the after effects or recovery from the outstanding pandemic in 1918.

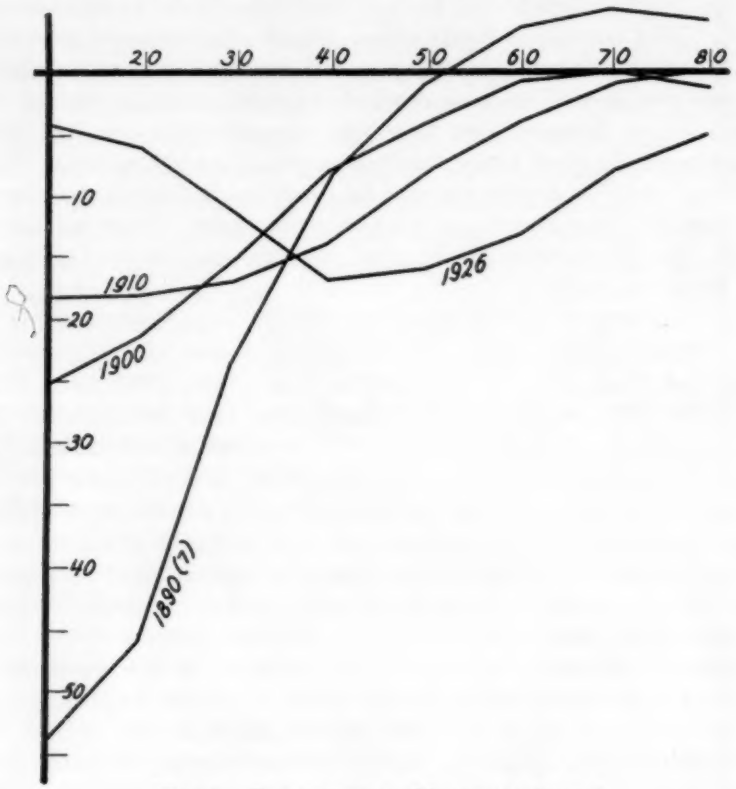

FIG. 1. EXPECTATIONS. MALES (10 STATES)

Attention is called to the following features common to the three sets of graphs for 1890, 1900, 1910 and 1920 given above:

1. At the early ages the trend of the expectation of life is definitely upward. That is, the expectation at these early ages is the lowest in 1890 and it increases, without exception, with each successive decennial year.

2. There is some age—near the prime of life—after which the order of the graphs becomes almost exactly reversed—that is, the trend of the expectation of

life becomes downward. The order would be exactly reversed if it were not for the abnormal year 1920.

In the next section many more graphs will be presented in each set and the intervals will be single years so that the important problem of

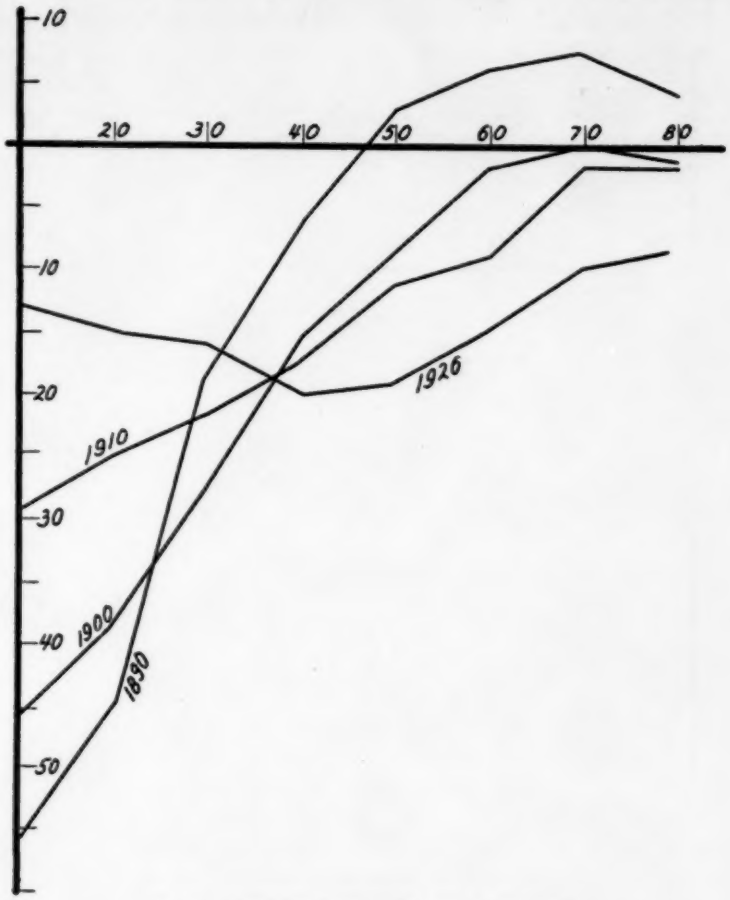

FIG. 2. EXPECTATIONS. MALES (7 STATES)

following the general trend will be confused more or less by the obtrusion of yearly fluctuations. There ought, therefore, to be no objections if we anticipate and accept the graphs for 1926 temporarily for comparison with these decennial graphs where there will be a minimum of confusion, and note their bearings upon the two features listed above.

The graphs for 1926 certainly corroborate the second feature and therefore add confidence to the belief in an ever falling expectation of life beyond the prime of life. In regard to the first feature, the male graphs for 1926 offer exceptions and would indicate that possibly the expectations at early ages are no longer increasing. On the other hand, it may well be argued that if 1920 is an abnormally "good" year the relative position of the base line is farther down, in which case

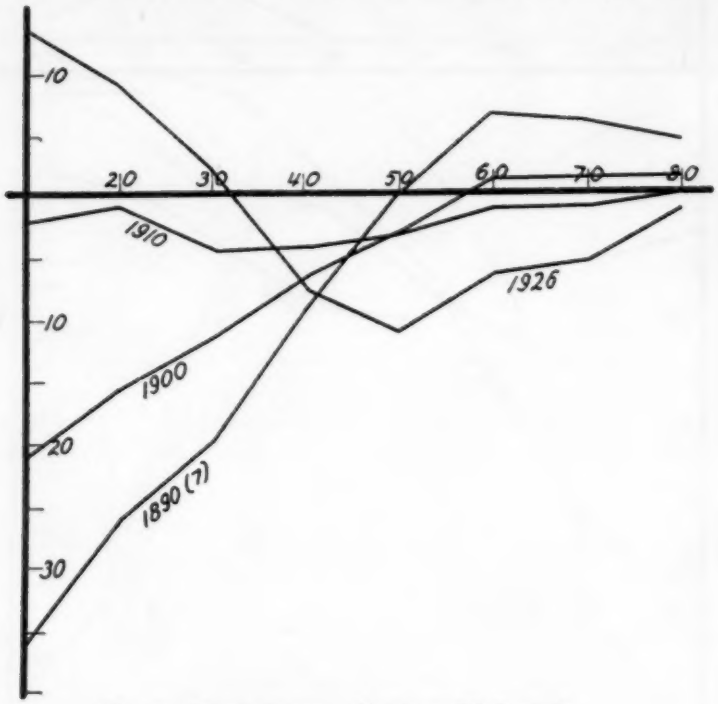

FIG. 3. EXPECTATIONS. FEMALES (10 STATES)

the male graphs might well extend above the base line at the earliest ages. While such an argument is quite sound and might lend weight to a belief that expectations are not decreasing—at least as yet—at the earliest ages, the writer believes that instead of the argument affording relief it really strengthens the theory that falling expectations are at least imminent at all ages. After all, the main question is whether the average length of life is beginning to decrease in this country and not the exact date at which the decrease begins. Let us examine more closely the nature of what we call the expectation of life.

The expectation of life at any age is simply the average length of life from that age on and is very much like the net value of the profits of the investments of an individual. The fact that he experiences a net loss does not deny the existence of some profits. To be concrete, we may be enjoying lower and lower death rates at early ages in this country—which is certainly the case—but the situation at later ages may be such that the expectation of life even at these early ages could very well be decreasing. A few readers of the previous article seem to have got the impression that that article claimed that *death rates* were increasing at all ages. That is not true and is not the point at all. No one questions the fact that death rates are still going down at early ages and although it is now just as clear that death rates have always been increasing at advanced ages, the improvements at the early ages have always overshadowed the losses at advanced ages and yielded a net gain in the average length over the whole of life—until possibly the present time, when the conditions at the advanced ages give signs of assuming the upper hand. It is entirely conceivable that the greatest gains in the saving of lives at early ages have already been made—even though the gains continue—and it has been fairly well established that losses at advanced ages are continually *increasing*. Now, the first natural sign that increasing losses at advanced ages are increasing their *relative* effect would be a regression of the age at which falling expectations begin and continued regression of that age must result ultimately in falling expectations at all ages. To refer back to the statement that the concession of the abnormality of 1920 may be a strong argument in favor of imminent falling expectations at all ages, we call attention first to the fact that that concession first strengthens the belief that expectations have always been falling at advanced ages, since the year 1920 was the one exception and since an appropriate adjustment of the graph for 1920 would remove it as an exception. Next we call attention for the first time to the female graphs in Figure 3. Not only would an appropriate lowering of the base line make the graphs unanimous in favor of a falling expectation at advanced ages but also the regression of the age at which these decreases begin from about 50 to about 30 would indicate that even though the females do not agree with the males in an indicated decrease in expectation at the earliest ages as yet, they do agree with the males that such a decrease is imminent.

Objections may well be made to the choice of the year 1926 to use in comparison with the decennial years, on the grounds that there was a

considerable amount of influenza in that year. This is true but in trying to select a year as a substitute for the next but lacking decennial year (1930) a choice had to be made between the last three years available—1926, 1927 and 1928—and while 1927 was not as "bad" as 1926, 1928 was worse and the use of the median seemed preferable.

Let us look a little further into the situation *since* 1920 and see whether any kind of trend is indicated among the recent years *taken alone*, and also see how much confidence should be placed in the graphs for 1926 which we have discussed.

THE PERIOD SINCE 1920

While we have no official data for populations since the census of 1920, we do have the annual mortality statistics. Let us examine the mortality statistics for males by ten-year age groups (5–14, 15–24, etc.) by successive years, and, as before, plot the excesses of these data over the corresponding data for 1920.

It should be perfectly understood that these graphs for males prove nothing as they stand, since, although the numbers of deaths are clearly increasing from about age 35 on, that is to be expected since the corresponding populations are also naturally increasing.

We should like to take this opportunity to shed further light upon the question whether 1920 is an abnormally "good" year, by including the mortality statistics of 1918.

While many will recall the situation in 1918 probably few realize that the increases in death rates among children were so great that even the general death rate was increased to what it had been even a quarter of a century before. It will be noted that 1918 was so disastrous (at early ages) that the graphs for the other years must now be plotted on a much smaller scale in order to include the graph of 1918. The graph of 1919 is also included to show not only the effects of the remaining cases of influenza but also a "drop" at advanced ages. It seems reasonable to suppose that abnormally high death rates at one period would naturally be followed by abnormally low death rates in the immediately following years—assuming no other factors to enter in—but it would not be deemed necessary to press this point if it were not for the outstanding severity in 1918 and its probably critical effect upon the very important year 1920. These graphs apparently check this theory and we would seem to be safe in regarding 1920 as an abnormally "good" year. The belief that expectations at

advanced ages have always been consistently downward in this country seems therefore, fairly well established.

We wish to take a moment to comment upon the use made occasionally in this investigation of the word "significant". For example,

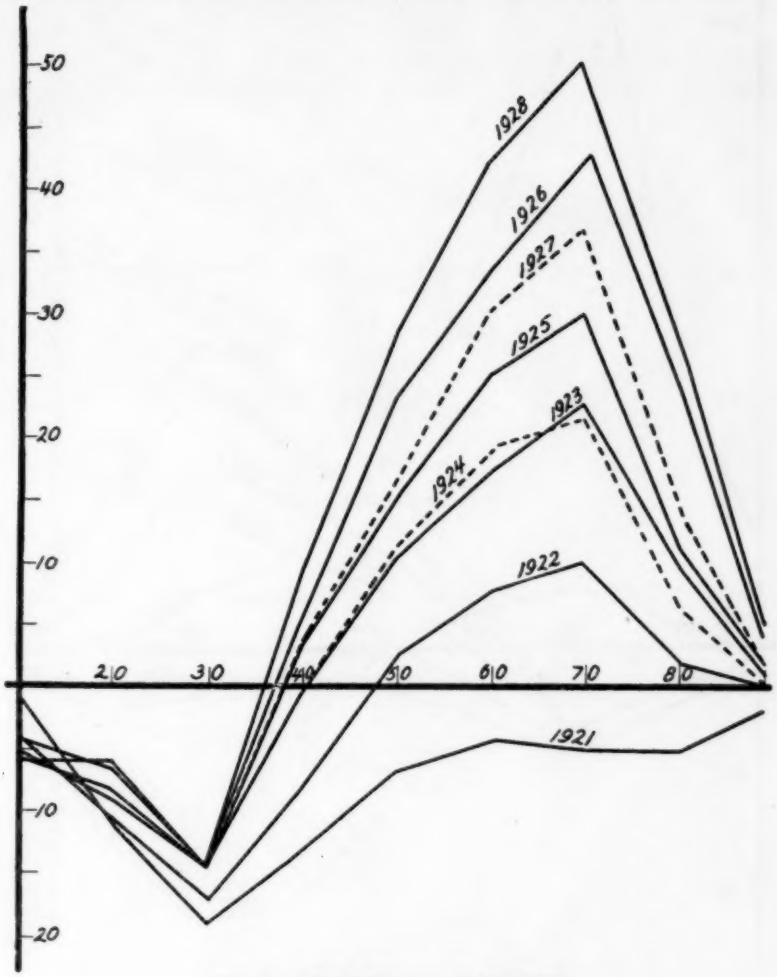

FIG. 4. MALE DEATHS (1920–1928)

a native of Japan wrote questioning a statement in the previous article to the effect that changes in death rates at the early ages during these successive individual years are not *significant*. He claimed that

if ratios instead of differences were plotted the changes would prove significant. We do not deny that the changes could be plotted so as

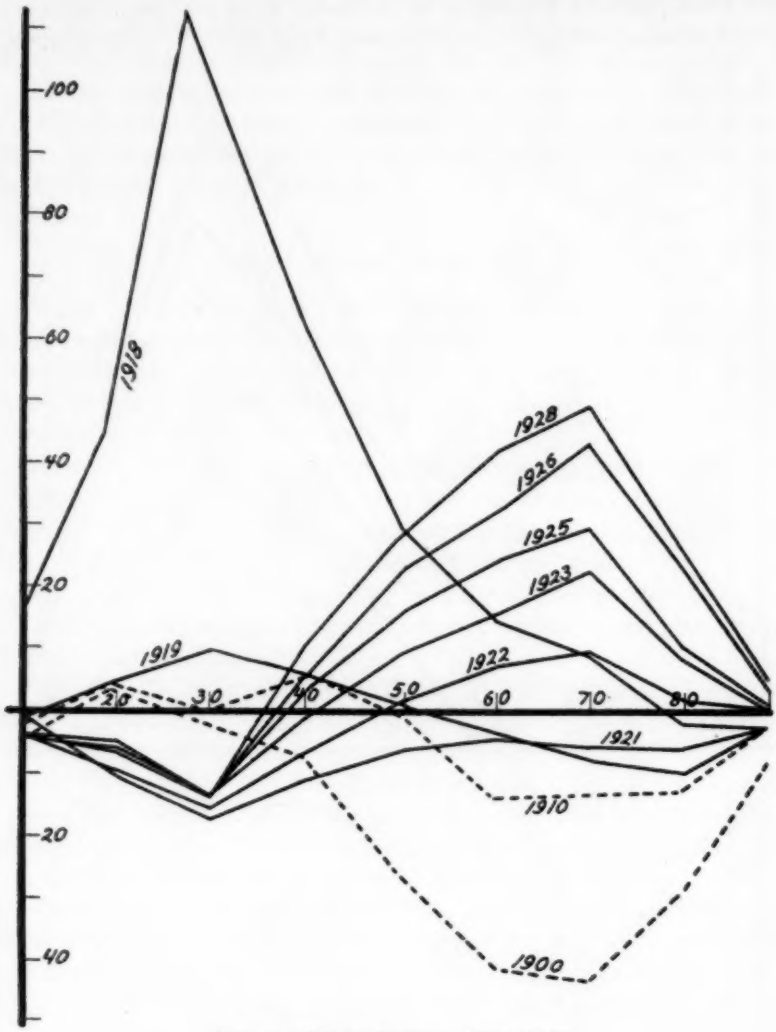

FIG. 5. MALE DEATHS (1918–1928)

to *appear large* but we are not interested in size here and no matter how large they were plotted they would not be significant in the sense used here unless the *direction* was clearly indicated. Even the data

of the graphs given above afford a good example. If the data for the age group 15–24 were arranged in order of size the years would rank 1921, 1922, 1927, 1923, 1925, 1924, and 1926; and for the age group 25–34 the ranking would be 1921, 1927, 1922, 1924, 1925, 1926, 1923.

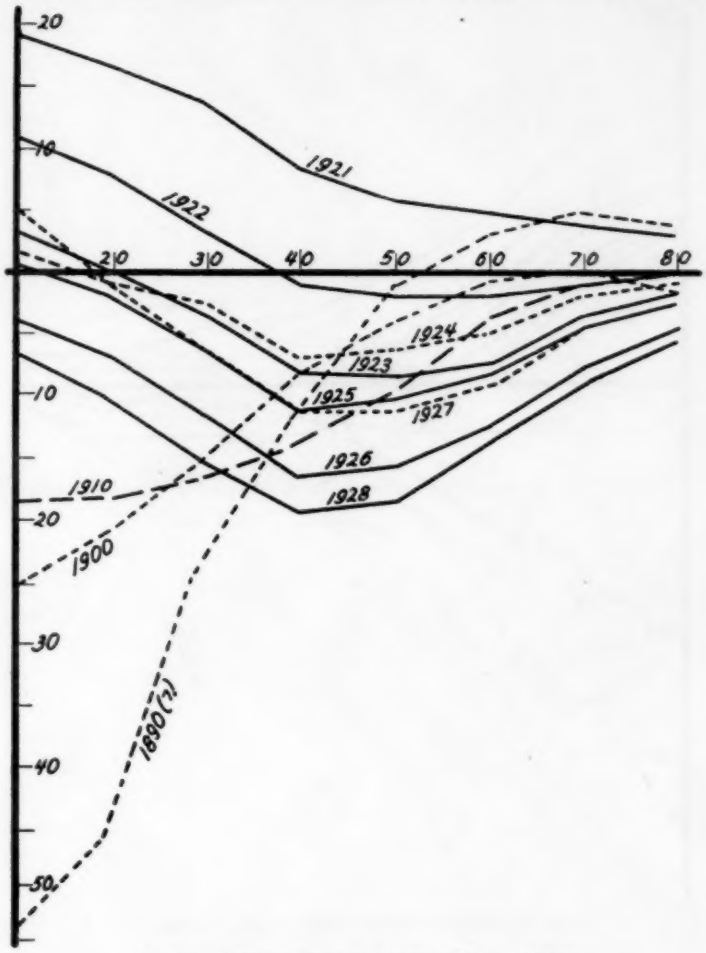

FIG. 6. EXPECTATIONS (1920–1928). MALES

In the light of other facts we could no doubt unravel some traces of trend here but nothing at all to compare with the situation at later ages. Taken alone, the writer would rather regard the changes at the early ages during the recent years as not significant.

To return to the mortality statistics for the individual years since 1920, it still remains to see whether the number of deaths are increasing faster than population or whether death rates are increasing. We must therefore have population data and having no official data the

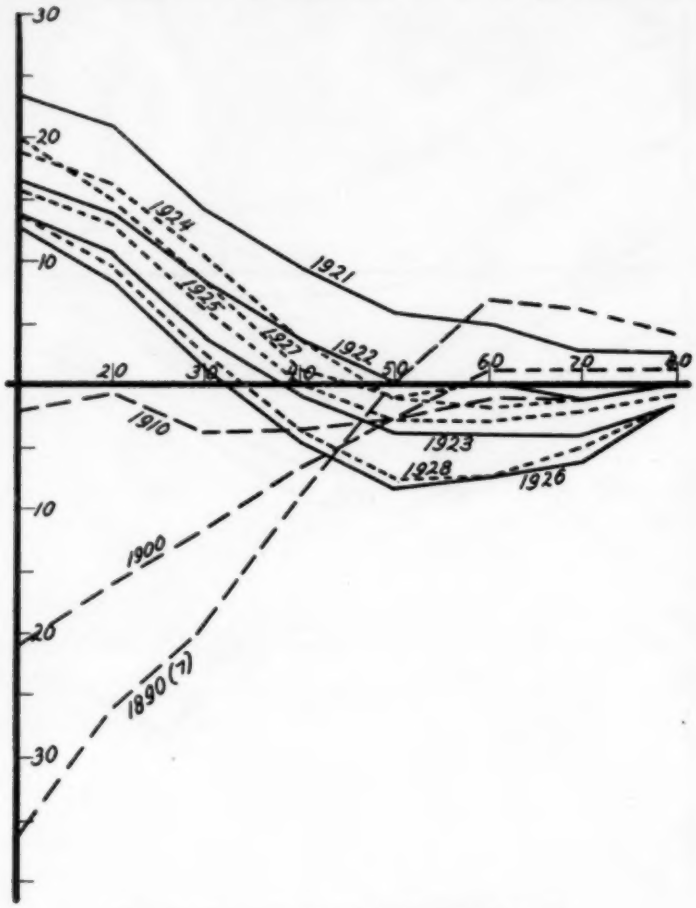

FIG. 7. EXPECTATIONS (1920–1928). FEMALES

total populations for the years 1921, 1922, . . . 1928 were extrapolated from the *total* populations for 1890, 1900, 1910 and 1920 for the ten original registration states, for each sex separately, and then these totals by years were distributed into ten-year groups exactly in the proportions existing in 1920.

The first sets of graphs are those of the expectations of life for females as well as males. In accordance with the theory that expectations are decreasing—at least at advanced ages—dotted curves are

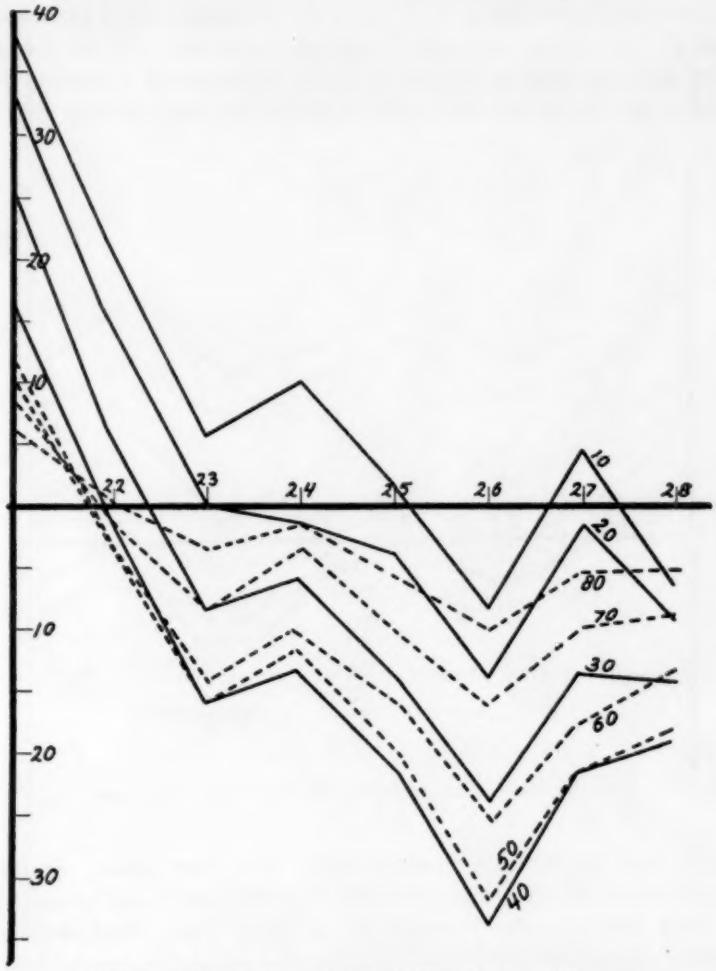

FIG. 8. EXPECTATIONS (1920–1928) BY AGE. MALES

used to refer to those years which do not follow exactly in the order indicated by the general trend. This scheme is employed merely to reduce possible confusion. It will be recalled that we were to expect

certain fluctuations when *annual* graphs were shown. Dotted curves of slightly different type are used also for the graphs of the preceding section corresponding to the earlier decennial years, which are included here for purposes of comparison.

These graphs would seem to speak for themselves and little can be added by discussion without becoming boresome. If we disregard for the moment any questions as to the methods of obtaining these graphs it would be interesting to examine the logic of any one who

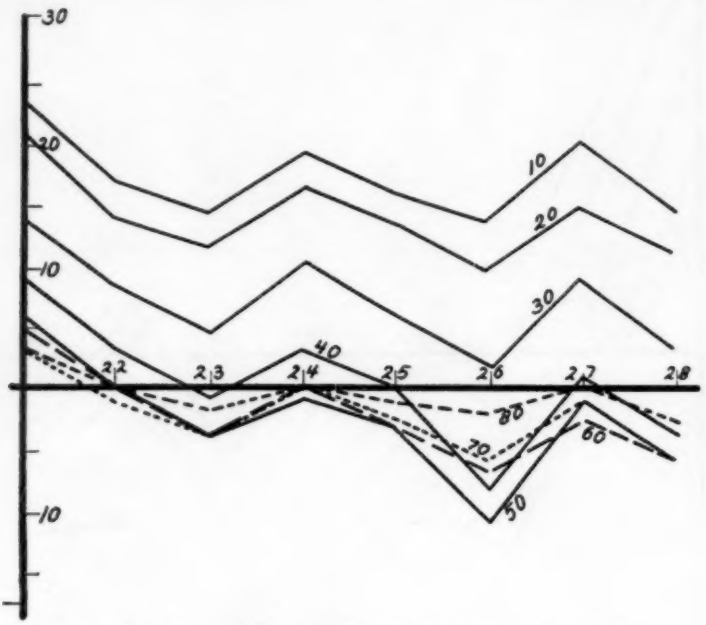

FIG. 9. EXPECTATIONS (1920–1928) BY AGE. FEMALES

believes that the trend is upward rather than downward. Even the exceptions to downward trend of 1924 and 1927 for the males and 1924, 1925 and 1927 for the females are no more than what we should naturally expect in the way of fluctuations when the results are presented by successive individual years and are not large enough to indicate the trend by themselves. The trend is not as smooth for the females as for the males, but those who are familiar with the usual statistical data for females will not be surprised at this and, after all, the curves themselves are rather remarkably smooth and definite.

Any decision that the trend of expectations is downward since 1920, for both sexes, is extremely important, since it checks with the analysis made independently for the period up to and including 1920 in the preceding section.

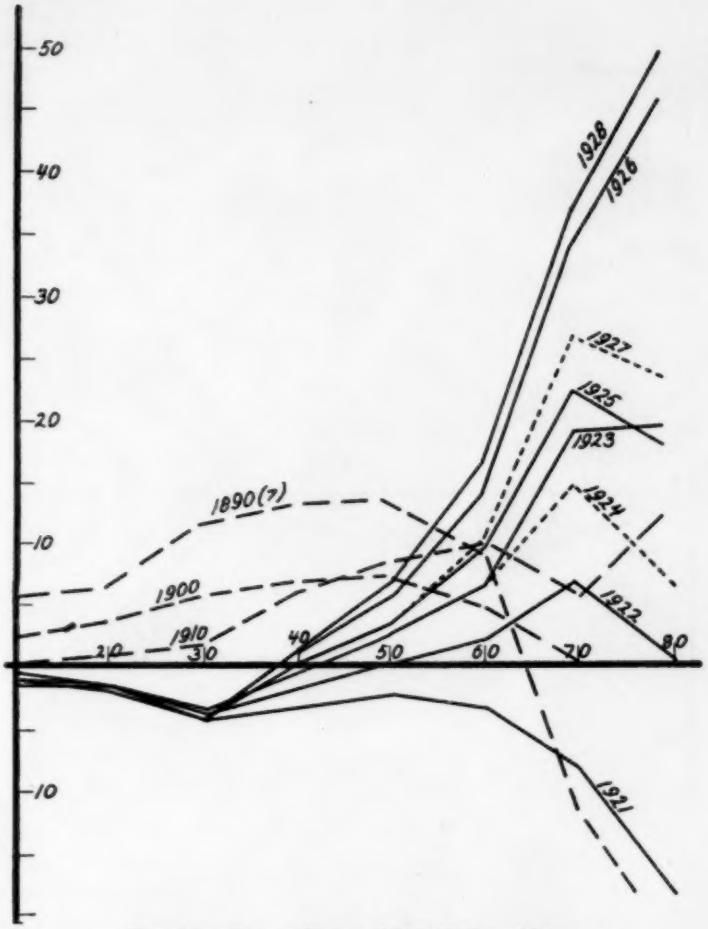

FIG. 10. DEATH RATES (1920–1928). MALES

Perhaps it will prove of interest to see the data of Figures 6 and 7 plotted in another way—by ages, instead of by years. This scheme is carried out in Figures 8 and 9.

In figures 10 and 11 are given the graphs of the death rates of the two sexes—or rather their deviations from the rates in 1920.

These graphs indicate that the trouble starts as far back as age 35 for the males and about 45 for the females, and that it is most significant for both sexes in the neighborhood of age 70. While the writer has his own individual ideas as to what are some of the factors or causes for this situation—which he expressed in the previous article—he feels that others are far better fitted for making this analysis and that it should be left to them.

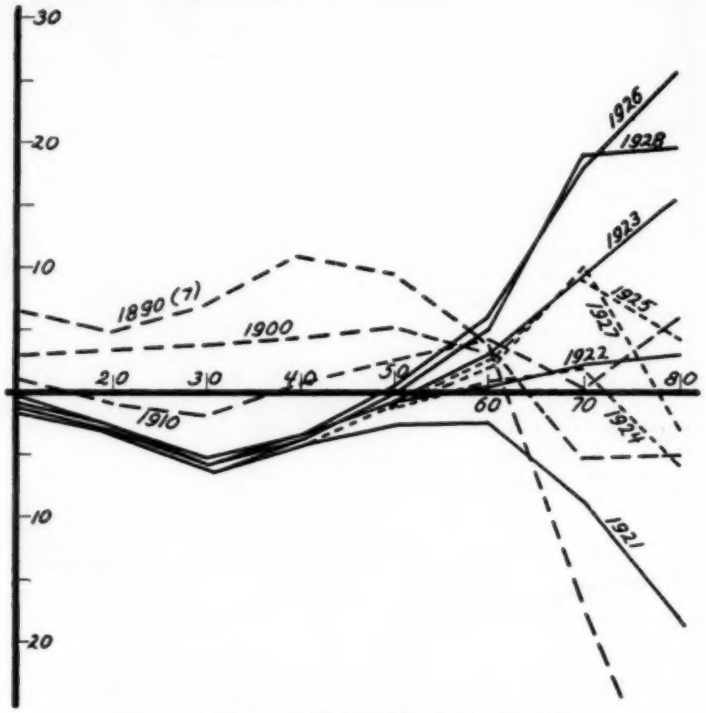

FIG. 11. DEATH RATES (1920–1928). FEMALES

AN IMPORTANT CRITICISM

There can be little argument over the plan of estimating the total populations for the individual years 1921–1928 by extrapolation. Some such method had to be used. There might, however, be differences in opinion as to what basic formula should be used, but until a better one has been proposed further comment seems unnecessary. On the other hand, the scheme of distributing these totals in ten-year age groups in the proportions existing in 1920 is open to some criticism.

The criticism was made by a correspondent after the appearance of the previous article and very reasonable arguments—which lack of space forbids to repeat—were offered for the feeling that 1920 would show abnormally low percentages of populations at advanced ages and that these low percentages would naturally be passed on—even in accumulated form—to the individual years following 1920 and therefore yield excessive death rates. The truth of this criticism could at least be examined and therefore had to be investigated.

The male populations by ten-year age groups (5–14, 15–24, etc.) of the ten original registration states for each of the censal years 1880, 1890, 1900, 1910 and 1920 were reduced to percentages to give the following results:

Distributions of Populations of the Ten Original Registration States by Percentages. Males

Ages	1880	1890	1900	1910	1920
5–14	21.6	19.8	19.1	17.9	18.5
15–24	19.5	19.5	18.0	18.7	16.1
25–34	15.5	17.0	17.4	17.5	17.3
35–44	12.3	12.6	14.0	14.4	14.6
45–54	9.1	9.4	9.6	10.4	11.3
55–64	6.0	6.0	6.2	6.2	6.9
65–74	3.2	3.5	3.4	3.5	3.5
75–84	1.2	1.3	1.3	1.2	1.2
85–94	0.2	0.2	0.2	0.2	0.2

If we use the percentages of 1880 as a base line the graphs of the deviations of the other years would appear as in Figure 12.

It will be noted that there has been a shift in the percentages away from the earlier ages, before age 25, to more advanced ages from 25 to 70, but that, contrary to the criticism referred to above, 1920 shows this shift. Therefore, there can be no criticism of the choice of 1920 *rather than any other decennial year* as a base, but it is clear that such a choice would not make any allowance for further shifts since 1920. In order to test the possible effects of such further shifts upon the results of this investigation a straight line was fitted to the percentages of the male populations of *each* ten-year age group for the five censal years considered above and the percentages and corresponding populations of the ten-year age groups of 1926 extrapolated. Not much can be said for the fittings obtained but as the

scheme seemed to lead, if anything, to excessive estimates of the shifts, the test would seem to have made every reasonable allowance. Then a mortality table was constructed for 1926 using the populations so determined. Figure 1—but now as Figure 13—is here repeated

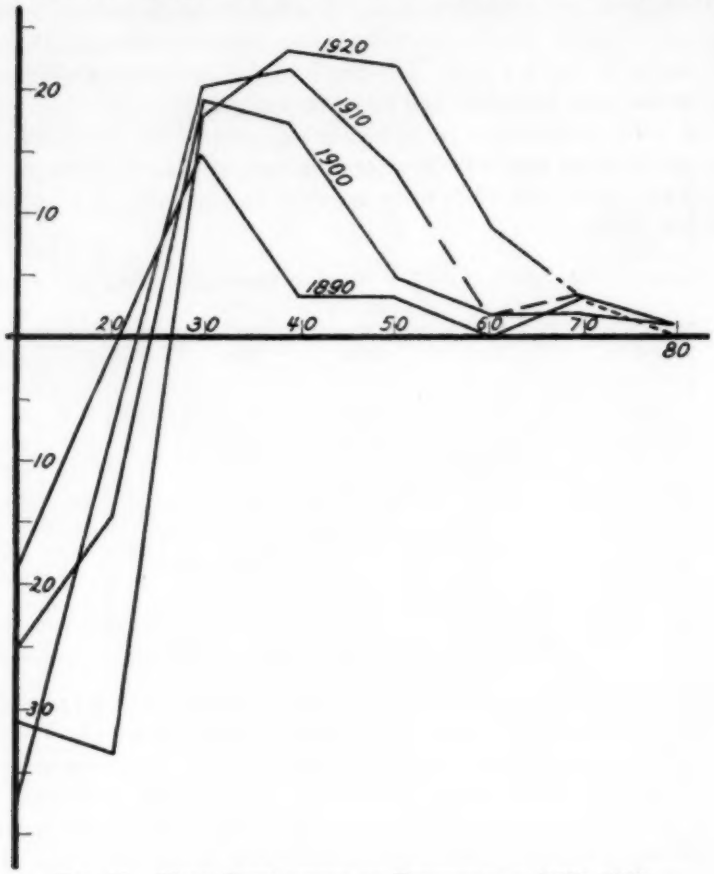

FIG. 12. MALE POPULATIONS BY PERCENTAGES (1880–1920)

but including the expectations of life for 1926 computed as explained above.

We believe that it will be agreed that although such an allowance does have an appreciable effect upon the graph of 1926 it has no appreciable effect upon the conclusion that the trend of expectations is now downward. It scarcely seems worth while to pursue the matter further without more accurate data.

WHAT TO EXPECT FROM THE 1930 CENSUS—AND SUMMARY

The results of the 1930 census will be very valuable in *helping* to substantiate or disprove the evidence offered in this article, since it will enable us to dispense with extrapolations of populations since 1920, but the writer wishes to express a warning against expecting conclusive results. Mortality tables constructed for individual years

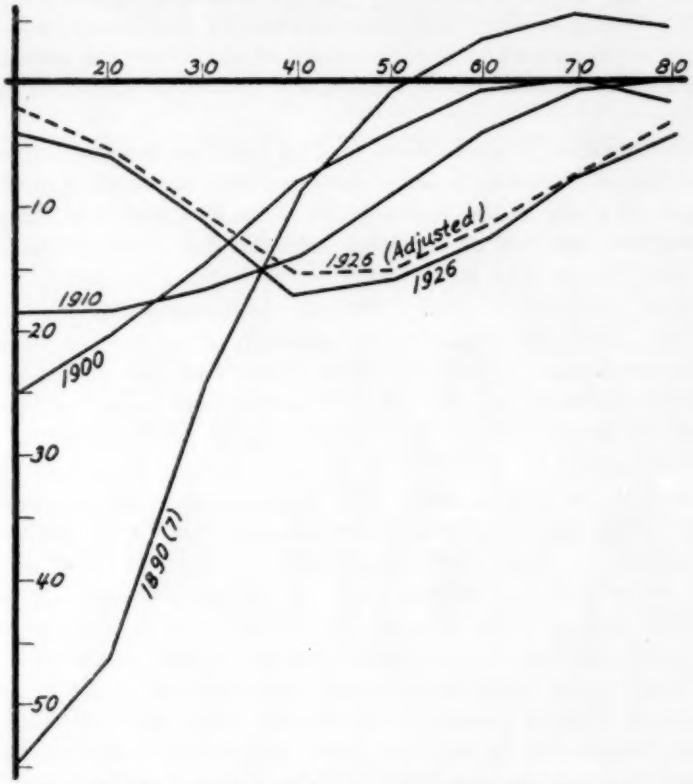

FIG. 13. ADJUSTED MALE EXPECTATIONS FOR 1926 COMPARED

and using mortality statistics of individual years must reflect mortality peculiarities of those years and will vary up and down from year to year regardless of whether the general trend is upward or downward. Hence, if, for example, there is an influenza epidemic in 1930, the number of deaths for that year is apt to prove excessive, in which case, the net result can not be regarded as necessarily substantiating the results of this article. On the other hand, there has been a consid-

erable amount of influenza in the last few years and the year 1930 may not only be unusually free from that disease but even show something of the recuperative effects which 1920 and 1921 seemed to show following the pandemic in 1918; in which case, the results for 1930 can not be regarded as conclusive in disproving the conclusions suggested here. The only way to analyze the situation is to watch the trend over as long a period as possible. Moreover, it is not a problem which will pass away and afford only this opportunity for solution; for, if the writer is at all correct in his own beliefs, we shall have the problem for a long time to come and the situation will grow more and more clear—and serious.

To summarize, it is admitted that we have too short an experience of a statistical character to draw absolutely sure conclusions of whither longevity is going in this country, let alone how fast it is going, but the evidence that we have would indicate that expectations of life have been dropping at advanced ages—or beyond the prime of life—ever since our first mortality records were started or even before and, also, that this situation has naturally developed to the point where expectations at all ages or at least from age 10 on may be beginning to decrease. This latter development seems particularly true of the males but the females seem to be not far behind. This downward trend in expectations is indicated in the period before 1920 and in the period after 1920 *independently*, and for each sex *independently*, and the trends of the two periods, for each of the sexes, "fit together". As far back as the middle of the year 1916 the writer stated, in an article in *Science*, that "individuals between the ages of 50 and 75 do not, on the average, live as long now as they did twenty years ago: and the extent of this retrogression is increasing". Then, a little over a year ago, the writer delivered a paper before a scientific meeting, in which he presented the first evidence that expectations at all ages might now be starting down—and at that time he had no evidence beyond the year 1925. Then, in the "previous article" in *Science*, referred to so frequently in this article, he presented further evidence that expectations might be starting to go down at all ages and included the evidence of 1926 and 1927. Finally, just as this article was about to be sent in to be published, the evidence of 1928 was received in manuscript form from Washington. The reader can see for himself that the results for 1928 merely "continue the story". A most important form of corroborating evidence is the results published by the Statistical Division of the Metropolitan Life Insurance

Expectation of Life

Males

AGE	1890*	1900	1910	1920	1921	1922	1923	1924	1925	1926	1927	1928
10	46.9	49.8	50.5	52.3	54.2	53.4	52.6	52.8	52.4	51.9	52.5	51.6
20	39.5	42.0	52.3	44.1	45.8	44.9	44.1	44.0	43.9	43.4	44.0	43.1
30	33.8	34.7	34.5	36.2	37.5	36.5	35.8	35.9	35.5	35.0	35.5	34.7
40	27.4	27.7	27.1	28.5	29.3	28.4	27.7	27.8	27.4	26.8	27.4	26.6
50	20.9	20.6	20.1	21.0	21.6	20.8	20.2	20.4	20.0	19.4	19.9	19.2
60	14.6	14.2	13.9	14.3	14.8	14.1	13.6	13.8	13.5	13.0	13.4	12.9
70	9.5	9.0	8.9	9.0	9.4	8.9	8.6	8.8	8.5	8.2	8.5	8.1
80	5.6	5.0	5.2	5.2	5.5	5.2	5.0	5.1	4.9	4.7	4.9	4.6

Females

10	49.3	50.8	52.7	52.9	55.2	54.6	54.3	54.8	54.5	54.2	54.9	54.3
20	41.9	43.0	44.5	44.6	46.7	46.0	45.7	46.2	45.9	45.5	46.1	45.7
30	34.9	35.7	36.5	36.9	38.3	37.7	37.3	37.9	37.5	37.1	37.7	37.2
40	28.4	28.6	28.9	29.3	30.2	29.6	29.2	29.6	29.3	28.8	29.4	28.9
50	21.8	21.5	21.5	21.8	22.4	21.8	21.4	21.7	21.5	21.0	21.7	21.1
60	15.6	15.0	14.8	14.9	15.4	14.9	14.5	14.9	14.6	14.2	14.7	14.2
70	10.1	9.6	9.4	9.5	9.8	9.4	9.1	9.5	9.3	8.9	9.4	9.0
80	5.9	5.6	5.5	5.5	5.8	5.5	5.3	5.5	5.4	5.3	5.5	5.2

Death Rates per 100,000

Males

10	52	40	32	30	28	26	25	25	25	24	23	25
20	71	60	48	45	35	36	38	38	36	37	35	37
30	106	84	70	62	45	46	47	47	46	46	44	46
40	133	108	106	84	70	75	81	81	84	86	83	87
50	183	159	163	132	122	134	141	142	145	154	145	158
60	309	293	315	276	263	285	302	302	312	328	316	340
70	556	606	630	605	575	633	678	663	695	739	712	753
80	1227	1323	1369	1319	1247	1322	1395	1344	1388	1499	1400	1512

Females

10	53	37	30	26	25	22	21	21	20	20	19	20
20	66	58	43	46	35	35	35	34	35	33	32	33
30	97	83	63	69	47	48	47	44	45	44	43	43
40	123	98	84	81	64	67	67	64	67	68	64	67
50	158	143	132	122	112	119	120	118	119	123	118	121
60	263	262	264	249	239	250	261	252	260	273	259	270
70	502	549	569	568	534	577	606	575	606	639	608	643
80	1127	1206	1250	1225	1153	1236	1288	1201	1242	1327	1215	1303

* Based upon seven states—instead of ten.

Company obtained from entirely different data—the experience of the insured lives of the company—and agreeing in all essential details with the results shown here. It can also be added that many more mortality tables were constructed by the writer than are represented here—all in a vain effort to find any evidence clearly contradictory to the conclusions suggested here. It would seem that about all has been done that can be done before the results of the 1930 Census are available. Whether the reader is convinced one way or the other—and the writer by no means feels sure—a substantial check surely has been offered against the wild predictions which have not infrequently been made in recent years of marvelous increases in the average length of life to be experienced in the immediate future.

DOUBTS AND SUSPICIONS CONCERNING CERTAIN FUNCTIONAL THEORIES OF PRIMATE EVOLUTION

BY EARNEST A. HOOTON

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"You are old," said the youth,
"and your jaws are too weak
For anything tougher than suet;
Yet you finished the goose,
with the bones and the beak—
Pray, how did you manage to do it?"

"In my youth," said his father,
"I took to the law,
And argued each case with my wife;
And the muscular strength,
which it gave to my jaw
Has lasted the rest of my life."

Alice's Adventures in Wonderland

It is tempting to rationalize concerning the causes of evolution. Science strives to explain evolutionary phenomena without recourse to the supernatural. It professes an utter incredulity of the miraculous. This is as should be. Faith is not knowledge. It may be possible to compound a satisfactory philosophy of life by mixing biology with metaphysics. Such a farrago may be spiritually efficacious but it is intellectually futile. It is like taking aspirin for a headache: the pain may be relieved but the cause remains intact and undiscovered.

On the other hand, there is almost as much danger in gulling one's self by plausible and orderly arrangement of biological phenomena in assumed relations of cause and effect, as in the narcotic habit of tincturing explanations of material facts with doses of the supernatural. It seems to me that students of evolution, in striving to avoid the Scylla of superstition, have not infrequently allowed themselves to be sucked into the Charybdis of fallacy. Such is too often the unconscious fate of the thorough-paced mechanist. Most writers on human evolution, however, are not so rash. Aiming at eclecticism, they

strive simultaneously to run with the Lamarckian hare and hunt with the Weismann hounds. Others take refuge in the covert of De Vries.

Now I must confess myself in complete accord with Bernard Shaw when he says a propos of Weismann's decaudalizing experiment:*

> . . . Weismann began to investigate the point by behaving like the butcher's wife in the old catch. He got a colony of mice, and cut off their tails. Then he waited to see whether their children would be born without tails. They were not, as Butler could have told him beforehand. He then cut off the children's tails, and waited to see whether the grandchildren would be born with at least rather short tails. They were not, as I could have told him beforehand. So with the patience and industry on which men of science pride themselves, he cut off the grandchildren's tails too, and waited, full of hope, for the birth of curtailed great-grandchildren. But their tails were quite up to the mark, as any fool could have told him beforehand.

But in spite of the futility of that particular experiment, a vast body of negative evidence against the inheritance of acquired modifications not only puts the modern Lamarckian on the defensive, but even stamps him as a biological apostate.

The situation is that no orthodox and self-respecting biologist can, in the face of the evidence, admit the transmission of acquired characters as a factor in evolution, and no kind of a biologist can give a satisfactory explanation of evolutionary phenomena without such an admission. Evolution by response of the organism to its environment and by hereditary perpetuation of such responses does not accord with experimental data; evolution by chance selection of combinations of characters–all inherent in the original germ plasm—puts more strain upon the credulity than the fantasies of primitive cosmology. We may look this difficulty squarely in the face and pass on.

I. FUNCTION AND PRIMATE EVOLUTION

I propose here briefly to examine certain functional theories bearing upon human evolution and upon primate evolution in general. Many of these theories have been presented so often that they seem to have acquired a certain measure of validity merely through repetition. They are all plausible and appealing in their apparent logicality. But their very simplicity and reasonableness arouses my suspicion.

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* Bernard Shaw. Back to Methusaleh. A Metabiological Pentateuch. Preface lvi, N. Y., 1922.

Arboreal life—its supposed effects on primate evolution

The first and most important of these functional theories has to do with the relation of primate cerebral evolution to the habits and bodily modifications assumed to have resulted from arboreal life. Such keen and competent anatomists as Professor G. Elliot Smith and Professor Frederic Wood-Jones have expounded most brilliantly and convincingly the dependence of neopallial growth upon arboreal habitat.* When our pre-primate ancestors, presumably primitive insectivores, took to the trees, they were already equipped with pentedactyle extremities including more or less opposable thumbs and great-toes— a heritage from even more lowly tetrapod precursors. In climbing up tree-trunks the hind-limbs of the animal were extended to bear the bodily weight, while the fore-limbs reached upward for new holds.

Thus began the "emancipation of the fore-limbs" and the specialization of the lower members for support—a process which was to be fraught with most numerous and far-reaching consequences. The first of these was the habit of squatting or sitting erect upon a branch or in the fork of a tree. The idle hands at the ends of the emancipated fore-limbs, having no new holds to reach for, got into mischief. They began to explore the more remote and less visible parts of the creature's own anatomy, thus creating a corporeal self-consciousness which in course of time reacted upon the neopallium, establishing sensory and motor areas in the cortical surface representing parts of the body previously unappreciated.

Equally important is the supposed effect upon the olfactory, auditory, and visual senses of the change from ground life to tree life. The small terrestrial quadruped is necessarily dependent upon the senses of smell and hearing, both in seeking food and in avoiding enemies. When, however, it climbs a tree, sight becomes far more important than smell or hearing. The visual areas of the neopallium increase enormously, whereas the olfactory bulbs of the archepallium remain stationary or decrease in relative size. The erect sitting or squatting position makes possible the free poise of the head upon the spine and facilitates the turning of the head so that the field of vision is greatly enlarged. The external ear tends to degenerate and to lose its mobility, because the animal now turns its head to catch sounds instead of cocking its ears. Even more astounding conse-

* G. Elliot Smith. Essays on the Evolution of Man, Oxford Press, 1924. F. Wood Jones. Arboreal Man, New York, 1916.

quences result from the free use of the hands for prehensile purposes. The primitive mammalian snout is in part a tactile organ. As Wood-Jones aptly says, the early terrestrial mammal "noses its way through life." Again, a grazing animal, with all four extremities devoted to support and locomotion, needs a snout, since the incisive or biting plane must be anterior to the visual plane. Otherwise the animal would get its eyes in its food and would be a blind and defenseless feeder.

The freed arms and opposable digits allow the beast to convey food to its mouth instead of bringing its snout to the food. The prehensile hands can pluck and perhaps tear into pieces of convenient size the comestibles. Thus the grazing function of the snout is transferred to the hands, and the former, through atrophy of function, begins to degenerate and to recede. The incisive plane shrinks back toward the visual plane: the canines, except when preserved for defensive purposes or for the piercing of tough fruits, undergo reduction in length and thickness.

The eyes in the primitive mammal are placed in laterally directed orbits, so that the fields of sight of the two eyes are separate; vision is monocular. But with snout recession the orbits are displaced from a lateral to a frontal position; the fields of sight begin to overlap: finally stereoscopic vision comes into being. With its poised and mobile head, its tactile and groping hands, and its perfected binocular vision, the animal gains a more complete knowledge of all parts of its body; it can see what each part is like and what it does. Hence, alongside of the areas of the neopallium devoted respectively to the sensory and motor functions of the various parts, there develop "pictured movement areas" which enable the animal to envisage, recall, and recreate desired motor and tactile situations. Rapidly increasing "association areas" occupy the regions of the cortex between the sensory-motor fields. Now the animal not only visualizes itself and recalls its movements, but remembering past actions and situations, anticipates the future and mentally creates new combinations, pleasing or displeasing. Ideational thinking has arrived. The only prerequisites of intellect are prehensile limbs and a convenient tree.

Some difficulties of the arboreal life argument

Of the lower primates, Tarsius is thought best to exemplify the evolutionary stage in which past experience and present bodily adaptations work together for good and lay open a humanward path of

evolution. We may now reflect a little upon this really seductive argument. It may be assumed, no doubt, that the same environment in the way of climate, habitat, food supply, et cetera, operating upon identical or similar animal forms, will effect in those animals identical or similar variations of an adaptive character. Thus a change from a terrestrial to an arboreal mode of life should have the same effect upon all similar pre-primates that took to the trees. Emancipation of the fore-limbs, development of an erect sitting posture, snout regression, and neopallial growth should proceed *pari passu* in all. But obviously such was not the case. The lemurs, for example, within the island of Madagascar alone, show a remarkable range and diversity of physical features, whereas they are generally arboreal and nocturnal and live upon essentially the same kind of mixed diet. The true Lemuridae, although generally arboreal, have shown little or no tendency toward emancipation of the fore-limbs, snout regression, displacement of the orbits from a lateral to a frontal plane, or any other of the "anthropoid" adaptations. I do not know whether they even assume habitually an erect sitting or squatting position. On the other hand, the Lorisidae have undergone both snout reduction and orbital displacement, but are, I believe, principally quadrupedal, and do not habitually assume an erect sitting or squatting position. The galagos also have somewhat reduced snouts and orbits displaced toward the frontal plane, but they are not exclusively arboreal. On the ground they are said to hop on their hind legs. Their muzzles are longer and more pointed than those of the lorises. Evidently either subtle environmental differences have brought about divergent adaptations in these various families of the suborder Lemuroidea, or the animals themselves are inherently different in their bodily organization and in their adaptive tendencies.

But it is hardly conceivable that the more or less uniform demands of arboreal life could have brought about such profoundly different adaptational responses in similar or identical animals. Therefore the divergently adapted animals must have been differently organized in order to be affected so variously by the same conditions of habitat. But then the entire argument for adaptation falls to the ground, since it is evident that the so-called "adaptive variations" are conditioned by the organism, or, at any rate, it is impossible to distinguish between a non-adaptive variation and one which is environmentally actuated and directed. If Tarsioid modifications are the result of arboreal life, why have not all arboreal lemurs undergone such changes?

Let us return to *Tarsius spectrum*. This animal, we are led to believe, anticipates in its adaptive modifications many or most of the possibilities of higher anthropoid development. The beneficial circle of action and reaction—neopallial growth promoted by manual exploration, and manual skill and dexterity accelerated by cortical representation—has already been established. What is the flaw in this perpetual evolution mechanism? Why has not Tarsius gone from strength to strength until it has transcended the heights of human achievement? "But" you say, "the existing family of Tarsius is conservative and has its evolutionary limitations. It has reached a stage of equilibrium." Well then, you are invoking hereditary and inherent differences in the original Tarsioid stock which destroy the force of the argument of the interdependence of manual freedom and neopallial growth.

Has the brain grown because of the emancipation of the hands or have the hands become emancipated because of the growth of the brain? If both, then the miraculous has been achieved. We eat our cake and we still have it. I do not believe that fumbling with the hands automatically develops the brain and increases the intelligence, nor do I think that a feeble brain can find other than mischief for idle hands to do. While writing this paper on board ship I have been watching a pet Cebus monkey entertaining himself nearby. I cannot conceive of any animal making a fuller use of its emancipated forelimbs and opposable thumbs and great-toes for exploratory purposes. Further this animal has an additional tactile and grasping organ in the form of a prehensile tail. With these immensely superior facilities for creating cortical representation why has not the Cebus monkey developed a larger neopallium and a more active mentality than man himself?

The alleged relationship between auditory degeneration and arboreal existence requires further demonstration. I suspect it to have been inferred from the reduction in size and mobility of certain primate ears, notably those of the orang-utan, the gorilla, and man. Does anyone possess accurate data as to the comparative acuteness of hearing in terrestrial mammals and in the arboreal primates? Or for that matter of the comparative olfactory keenness of these animal groups of different habitat? I do not believe that the external ear in man and the anthropoids has been reduced in consequence of the increased facility with which the poised head may be turned to catch the sound waves. The orang and the gorilla have very small and probably

degenerate ears, while the chimpanzee has fine, large aural appendages "projecting", as Sir Arthur Keith once remarked, "like the port-sails of a steamer in the tropics." But orangs and gorillas have more massive and overbalanced jaws than have chimpanzees and consequently should have less facility in rotating their heads, because of the higher hafting on the occiput of their more powerful nuchal musculature. If size and mobility of the external ear is a criterion of auditory acuity, the chimpanzee should hear at least twice as well as the gorilla or the orang-utan. Yet the chimpanzee is far more arboreal than the gorilla and, consequently, according to hypothesis, should have less need of a keen auditory sense. Man's head is more nearly balanced on his spine than that of the orang-utan or the gorilla, so that presumably it may be rotated more easily to catch sound waves. Yet the human ear is much larger and less degenerate than that of either of these anthropoids. Finally Tarsius, which can rotate its head almost 180°, has, perhaps relatively the largest and most mobile ears of all the primate order.

The correlation between snout reduction and frontal position of the orbits resulting in stereoscopic vision is another dubious deduction. The baboon, which has the largest and most projecting snout of all primates, has anteriorly directed orbits, and, presumably, stereoscopic vision. The shortest snouts below man are found in the New World monkeys and marmosets, but their orbits are not completely frontal in position as those of the long-snouted baboons and macaques. Of course it may be argued that the baboons have redeveloped snouts as a result of a secondary adaptation to pronograde life on the ground, while retaining the obvious advantage of stereoscopic vision. But this can scarcely apply in the case of the Semnopithecinae and many other essentially arboreal Old World monkeys which have much longer snouts and more frontally directed orbits than the Cebidae.

Brain size and jaw protrusion

Perhaps the most striking and plausible of all Lamarckian theories of primate evolution is that which relates the increase of brain size to the reduction of jaw protrusion through decrease of the temporal muscle area of attachment to the skull vault and consequent elimination of a compressing and growth-inhibiting effect. Of the application of this theory to the humanoid phase of primate evolution, I shall have something to say later. Here I wish to remark only upon its validity

as an explanation of facial and brain-case proportions among the lower primates and the anthropoid apes.

I do not know that anyone has demonstrated by any metric or statistical method that there is any constant relationship in the various groups of the primates between snout protrusion, cranial capacity, and proportion of the skull vault covered by the temporal muscles. A casual survey of primate crania leaves me with the following impressions:

(*a*) While many of the Lemuroidea exhibit a low mammalian status in their great snout protrusion and small development of the brain-case, others, and notably the lorises and galagos, show marked snout reduction, and the area of the skull vault covered by the temporal muscle attachments varies widely and seems to be independant of the length of the snout. Many lemuroids seem to have relatively smaller areas of the skull vault devoted to temporal attachments than have some of the Anthropoidea, notably the Cercopithecidae and the Simiidae.

(*b*) In Tarsius the snout is much reduced; the brain case is enlarged, and the area of temporal attachments to the cranial vault is comparatively small. But in the marmosets (Hapalidae), snout reduction has proceeded much farther without a corresponding increase in brain size. Yet some of the marmosets have temporal muscle attachments covering almost all of the cranial vault. If the reduction of the snout has anything to do with the prehensile use of the hands it is hard to see how the Hapalidae fit into the picture, since these animals have rudimentary thumbs and digits provided with nails instead of claws. Their hands are non-prehensile. Their brains are in many respects inferior in development to those of the lemurs.

(*c*) The Cebidae (American monkeys) have in general more protrusive snouts than the Hapalidae, but their brains are relatively and absolutely larger than those of the marmosets and of much superior development. In general the areas of the attachment of the temporal muscles to the cranial vault are relatively smaller than in the marmosets.

(*d*) The Cercopithecidae are extremely variable in this relationship of snout projection to temporal muscle area, but in general they show a lower evolutionary status than the admittedly inferior Cebidae. Of all primates the baboon (Cynocephalus) shows the most disproportionately large and protruding snout and the relatively smallest brain-case. But although the cranial vault of this animal is almost completely devoted to the attachments of temporal and nuchal mus-

culature, it does not show the exaggerated development of muscular scaffolding in the shape of sagittal and transverse crests such as are found in the adult male gorilla and orang-utan. Nor does the baboon show the tremendous post-orbital constriction of the frontal bone which is a characteristic feature of the skulls of the above mentioned apes.

(*e*) Among the Simiidae or anthropoid apes the gibbon has the longest canines and a facial protrusion equal if not superior to that of any of the larger apes. But the relative area of temporal muscle attachment to the cranial vault is much less in the gibbon than in any of the large anthropoid apes and its brain-case shows fewer signs of having been moulded or modified in form by muscular pressure. The snouts of the gorilla and the orang, while markedly projecting, seem to offer insufficient mechanical justification for their huge cranial crests and exaggeratedly large areas of temporal attachment to the cranial vault. Probably these crests are to be regarded as secondary sexual characters and, to some extent, as old age features, rather than essentially adaptive and structurally necessary features of their cranial architecture.

In general it would appear that the association of snout projection and temporal muscle area with form and size of the brain-case exhibit among the various groups of infra-human primates no such consistency of interdependence as would justify the assumption that a correlation between the form and size of these parts in fact exists.

Finally the study of the growth of the skull in anthropoid apes—as well as in human beings—clearly shows that the brain and the brain-case attain virtually their adult size before the accelerated growth of the jaws sets in. It is well known that the temporal crests of the young male gorilla ascend the parietals almost *pari passu* with the eruption of the permanent dentition and that the formation of the sagittal and occipital crests does not occur until the canines (the last teeth to erupt) have been cut. But it is my contention that this interesting association of growth processes can have no constricting effect upon brain development, since in all probability the growth of the latter has practically ceased before the commencement of the jaw and temporal muscle changes in question.

Unfortunately an almost complete lack of precise metrical and statistical information on the subjects discussed above leaves the entire matter open for debate and speculation. One may admit that some sort of general compensatory relationship between jaw size and

brain size obtains in primates and lower mammals. It is conceivable that the hyperfunctioning and consequently excessive nutritive demands of some part of the organism, such as the masticatory apparatus, may be detrimental to the development of adjacent parts, such as the brain. Loosely speaking, the generalization "the more jaw, the less brain" has a certain validity when applied to the primates. Nevertheless I do not believe that jaw reduction and decreased area of temporal muscle attachment in themselves effect or even promote cerebral expansion and growth. Such atrophy of the masticatory apparatus may, indeed, release for other needs a certain surplus of nutritive material or growth energy.

Erect posture as a result of arboreal life

It seems to me that, on the whole, the most satisfactory functional theories pertaining to anthropoid and human evolution are those which explain man's present posture and gait in the light of a previous arboreal habitat, and which postulate as prerequisites of erect biped walking the brachiating method of locomotion in the trees, and the erect sitting and climbing posture exhibited by the present anthropoid apes. There can be no reasonable doubt of the substantial truth of the assumption that certain visceral, muscular, and osseous features in man and the anthropoids are the results of adjustments brought about by arboreal arm suspension and by the habit of sitting erect. That this new vertical position of the trunk was not an inevitable consequence of an increase in size, whereby the animal was restricted in climbing to the larger branches and to the vertical tree trunk, is evidenced by the complete attainment of all of these modifications by the gibbon, which is inferior in size and weight to many pronograde monkeys. It is almost impossible to avoid the conclusion that the elongation of the upper extremity in this animal, as in the great anthropoids, is a direct consequence of its brachiating method of locomotion. We may dismiss as a biological enigma the mechanism whereby such an adaptation has been transmitted. But if the arms of anthropoid apes have become lengthened by use in suspension and locomotion, it does not necessarily follow that their legs have been shortened through atrophy and loss of function. Indeed the gibbon, which has by far the longest arms relative to its trunk length, has also proportionately the longest legs, and this in spite of the fact that it is the brachiator *par excellence*. Similarly, among the New World Monkeys, Ateles (the spider monkey) has the longest

arms and is most addicted to brachiation, while retaining at the same time long legs and a remarkably elongated and prehensile tail. It therefore seems that, as regards the limbs, no principle of correlated or compensating adaptations can be invoked.

Again, we may regard the gorilla as possessed of pelvic and pedal structure best adapted among apes for bearing the weight of the body upon the lower extremities in an erect or semi-erect posture. In this animal the iliac blades are greatly expanded transversely and are slightly recurved anteriorly so as to create some semblance of iliac fossae. There is even some foreshadowing of an ischiatic notch, formed by the incipient downward bending of the posterior portions of the ilia adjacent to the sacro-iliac articulation. The pelvis of the orang-utan is not so human in some of these features, that of the chimpanzee even less so, while the pelvis of the gibbon in its great height, its elongation of the iliac blades in the ischiatic axis, and in its lack of transverse development of the ilia, closely approximates to the pelvic type of the pronograde monkeys.

Corresponding to its pelvic morphology, the foot of the gorilla is of all anthropoid feet relatively the broadest, with the most elongated heel, the best developed great toe, and the most pronounced reduction of the four outer digits. It seems to approach most closely of all prehensile primate feet to the status of a weight supporting organ. The gorilla alone of all anthropoids is said to apply the sole of the foot flat to the ground in walking. The other apes walk on partially clenched feet with the lesser digits flexed toward the hallux and with the weight resting largely on the outer border of the sole. The foot of the chimpanzee is a frankly prehensile foot, in all respects less human than that of the gorilla. That of the orang-utan is very long and narrow; it has a small, degenerate, set-back hallux, lacking the nail and sometimes the last phalanx. Its outer digits are hooked or recurved, enormously elongated, and presumably adapted to the animal's habit of hanging from its feet. The gibbon's foot is even more elongated, narrower, and apparently more specialized for prehension. Except for its long legs, then, the gibbon, of all the anthropoids, is obviously least adapted for erect posture and bipedal gait. Yet the gibbon is the only ape which habitually walks erect when on the ground. Of course it may be that this posture and gait are forced upon the gibbon by the excessive length of its arms, fore-and hind-limbs of approximately equal length being desirable for efficient quadrupedal progression. Similarly man progresses only awkwardly on all fours,

since in his case the legs are too long and the arms are too short. Hence we crawl on hands and *knees*. But the gibbon could, presumably, flex its trunk on its thighs, bend its arms at the elbows and thus move on all fours. However it does not choose to crawl.

The gorilla, in spite of his supposed weight-bearing adaptations, is said to be habitually quadrupedal when on the ground. But when he wants to stand erect and walk a few steps, he does. The chimpanzee commonly uses all four extremities in walking or running on the ground, but he can and does stand and walk erect, when he chooses to do so. Evidently then, within certain limits prescribed by the degree of specialization of appendages, the posture and gait of anthropoids are determined by choice and habit rather than by structural adaptation. Nor do the animals apparently best adapted, or most nearly adapted, for bipedal progression necessarily adopt that gait when on the ground. Probably gorillas, chimpanzees, and orang-utans never walk along the branches of trees on all fours like pronograde monkeys. Such a method of locomotion would involve them in difficulties of balance which would make it impracticable. But these anthropoids, erect in the trees, are pronograde on the ground.

Man shows obvious and rigorous adaptations of the pelvis and the lower limbs to an erect posture and a bipedal gait on the ground. Yet nothing in the structure or habits of existing anthropoids justifies the inference that human gait was dictated or prescribed by arboreal adaptations of our prehuman ancestors. It may be asserted that erect posture in the trees and possibly the use of the fore-limbs in brachiation made possible and foreshadowed the erect posture and biped gait of terrestrial man, but they did not impose upon him such posture and gait. The elongation of the human legs and the modifications of the human pelvis and feet so suitable for upright progression were probably subsequent to the habit of erect ground walking.

As stated previously, I have no serious criticism to bring against the theory which derives the essential features of man's posture and gait from previous arboreal habits and adaptations. That the human foot has been modified from a prehensile anthropoid type seems to me to be one of the most firmly grounded inferences of biological science and is probably conclusive evidence of a previously arboreal habitat. (Incidentally, the absence in the human foetus of a convex cunieform joint for the first metatarsal does not trouble me in the least. Recapitulation is a coarse-meshed sieve, which retains only a few obvious ancestral features.) But the most cogent argument for a tree-dwelling,

anthropoid derivation of man lies not in the similarity of the human foot to the prehensile primate foot (as a matter of fact they are markedly different), but in the general body of evidence of morphological relationship, amounting almost to identity, which man manifests toward the rest of the primates which are arboreal.

The recent theory that man has sprung from a small, ground-dwelling, Oligocene primate, seems to me to have nothing whatever to commend it. It has been refuted, in my opinion, completely.

Here I may refer briefly to the evidence from similar foetal proportions of divergent growth in man and the anthropoids admirably set forth in various studies by Professor Adolph Schultz. The thumb, for example, in both foetal orang-utan and in the foetal human being, is relatively larger than in the adults of either species. But in the course of post-natal growth the thumbs of both man and the orang become markedly reduced in size, a process which affects the ape to a greater degree than the human primate. It seems to me that such precise embryological data, based upon metric observations, when considered in conjunction with the careful study of the actual motor habits of live animals, offer perhaps the most convincing class of evidence in favor of evolution by functional adaptation. But even in the example cited we cannot be certain whether the orang uses his fingers to the exclusion of his thumb because the thumb is too short, or whether the thumb has degenerated through lack of use.

The loss of the tail

The disappearance of the external tail in the anthropoid apes and man has given rise to various speculations, some of which attribute its loss to atrophy through disuse. The best known of these is probably that advanced by Darwin, who called attention to the calloused and abraded stump of the caudal remnant in certain macaques, observing that the animal in sitting upon its haunches would chafe and rub its short tail, which would necessarily be bent underneath to avoid being pinched between the ischial callosities and the ground, or would be extended straight out behind. In either case it would be subjected to a good deal of friction. In his opinion the tail may well have disappeared through attrition, the blood and nerve supply having been cut off. This reminds me of the Wayao story of the Pig and the Baboon. The pig and the baboon were sitting on a hillside one very chilly day, when the baboon remarked: "Isn't the wind cold? It's enough to wear the end of one's nose down to a blunt snout." "Yes"

replied the pig, "it's really enough to blow the hairs off one's buttocks and leave a bare dry callous patch." "Look here," said the baboon, getting cross, "you are not to make personal remarks!" After that the pig and the baboon were no longer friends.

It seems probable that the disappearance of the tail was involved in the changes which took place in the evolution of the small gibbonoid type of anthropoid, when the rod-like lumbar column of the pronograde springing primates was reduced by one segment through the sacralization of the last lumbar vertebra and the shifting cranialwards of the pelvic girdle. This theory of the pelvic shift, originally proposed by Rosenberg, and ably defended by Keith, maintains that the loss of vertebral segments takes place only at the tail end through the sacralization of lumbar vertebrae, the transmutation of sacral into caudal segments, and the ultimate "shucking off" of the terminal segments of the coccyx. In man we are informed that this process of disappearance of coccygeal vertebrae has been arrested by the fact that the coccyx has been bent underneath and the tail muscles spread out like a hammock, thus performing a new function in forming the floor of the pelvic aperture and preventing the viscera from sagging downward. This theory does not lack ingenuity and may well be correct in so far as it concerns the alteration in function of the remaining caudal vertebrae, but it does not explain the disappearance of the external tail. It is not enough to point out that for giant primates like the great apes and man a tail is neither useful for prehension as in the Cebidae, nor for a balancing rod as among many pronograde Old World monkeys, nor yet for a portable "comforter" or bed-spread as employed by some lemurs. Loss of utility and atrophy of function do not necessarily lead to elimination of the disused part. Nature carries a good deal of excess baggage in many animal organisms. Of what use is the elephant's tail?

In this connection it seems that insufficient consideration has been given to the fact that some of the lemurs, notably the Lorisidae, are practically devoid of external tails. It is not impossible that man may have descended from a primate stock which was tailless, even in the lemuroid stage of evolution, although I do not think that this is probable.

In the last analysis we can state only that man's remote ancestors must have had tails and that these were lost, in all probability, prior to, or simultaneously with, the small anthropoid stage of evolution. Doubtless a short stumpy tail, such as that possessed by some

macaques, would be of little use either to a brachiating arboreal primate or to a terrestrial bipedal primate. But it seems to me that an active prehensile tail, such as that appended to the spider monkey, might be very handy indeed, even to man, provided it were not so long that it could be trodden under foot.

II. ALLEGED FUNCTIONAL FACTORS IN RACIAL DIFFERENTIATION

It is commonly and perhaps correctly assumed that many of the physical features which distinguish different groups of mankind have originated as the result of functional responses of the body to various environmental stimuli and have, in some way, been stabilized and transmitted as hereditary characters in the several human races. Perhaps it may be worth our while to examine critically some of the more popular of these adaptive theories as currently applied to the problem of racial differentiation.

The brain and the jaws

One of the most widely disseminated of these beliefs is that which connects extreme narrow-headedness with masticatory hypertrophy, especially of the temporal muscles, or with facial protrusion (prognathism). The Eskimo is the most notorious example of this assumed relationship between dolichocephaly and over-developed chewing muscles. "Typical" Eskimo crania are well known to exhibit very elongated, narrow, and scaphoid skull vaults, with remarkably extensive temporal muscle attachments and high lying temporal crests, to which are joined facial skeletons of remarkable breadth, with massive frontally and laterally projecting zygomata and malars, broad palates, strong teeth, and heavy mandibles. The masseter attachments on the inferior surfaces of the malars and zygomatic arches are extraordinarily extensive; the gonial angles of the mandible are everted and the internal pterygoid attachments are large and rough; the ascending rami are broad and low; the lingual faces of the horizontal rami often show an irregular thickening of compact bony tissue which has been called the mandibular torus. Altogether it is difficult to avoid the conclusion that the facial skeleton of the Eskimo shows marked evidence of an exceptionally vigorous habit of mastication. The condition of the teeth in Eskimo crania generally supports this assumption.

The theory is that the great temporal muscles of Eskimo compress the side walls of the cranial vault, restricting the lateral growth of the

brain and the brain-case; hence, by way of compensation the brain and the skull elongate antero-posteriorly, producing the characteristic dolichocephaly and leptocephaly. The sagittal thickening of the skull roof in the median line, producing the scaphoid appearance, is explicable, according to this view, by the supposition that the medialward pressure of the temporal muscles stimulates bone growth along the sagittal edges of the parietals, thus providing a reinforcement against the strains incident to chewing.

These adaptations, resulting in such a profound modification of cranial and facial form, are all ascribed to the unusual demands upon their chewing apparatus which Eskimos make. We are told that these people live almost exclusively upon a diet of flesh and fish, mostly of a formidable toughness. More than this, the Eskimos prepare skins for clothing by chewing them; they exercise their jaws upon walrus skin just as the vulgar in our own society masticate the gum of the chiclé tree. Finally the Eskimo is said to make use of his teeth for the untieing of frozen knots and lashings and in a variety of other vigorous and unusual ways. So that it is altogether natural to conclude that the Eskimo owes his cranial peculiarities to his diet and dental hypertrophy. I was brought up on this theory and for a long time I accepted it implicitly. I have even attempted some modest amplifications, based upon my own observations.*

Alas! My childish faith has been mangled by horrid, gnawing doubts. The sad process of disillusionment was as follows. Firstly, it is matter of common observation that the typically Eskimo features (i.e., dolicho-leptooscaphocephaly with euryprosopy) are much better developed in males than in females. But the practise of chewing skins to prepare them for clothing and of softening frozen boots by mastication is limited to the women! Yet the men have broader faces and their skulls seem to show clear evidence of greater development of the masticatory apparatus.

Again, I had supposed that frozen raw fish and meat would be exceptionally tough and stringy and would require a great deal of chewing. But Vilhjalmur Stefansson, who has lived exclusively on such a diet for long periods of time, informs me that the process of freezing and melting breaks up the fibres of the fish and reduces it to a consistency much like that of ice cream, so that it can be swallowed with very little mastication. He is of the opinion that Eskimo food by no means requires strong teeth and great jaw muscles.

* Hooton, E. A. On certain Eskimoid characters in Icelandic skulls. *Am. Journ. Phys. Anthrop.* Vol. I, No. 1, pp. 53–76, 1918.

Further, I seriously doubt whether the temporal muscles in chewing exert much pressure inwards against the walls of the cranial vault. Most of the strain must be vertical or nearly so. Rotary chewing movements so characteristic of vigorous mastication, do indeed involve the temporal muscles, not simultaneously but alternately. It seems probable that the pterygoid muscles and the masseters are much more important in lateral chewing movements. The temporal muscle area is the thinnest part of the cranial vault. It is the only part of the vault which exhibits externally the swellings of the cerebral convolutions. It is improbable that the action of the temporal muscle exerts much medialward pressure; otherwise these external markings of the convolutions would not be in evidence.

The correlation of temporal muscle area and cranial characters

In order to test out the hypothesis which relates the size of the temporal muscle area to dolichocephaly and to the form of the head in Eskimos, I have undertaken certain experiments, details of which will be published in another paper. The areas of the temporal muscle attachments to the skull vault were calculated for each skull by means of a planimeter; the combined areas were then correlated with cranial capacity, head length, head breadth, length-breadth index, and with other measurements and indices.

If the enlarged temporal areas of attachment in the Eskimo tend to restrict brain growth we might expect a negative correlation between temporal muscle area and cranial capacity: i.e., the larger the temporal muscle area the smaller the capacity. There is on the contrary a high positive correlation between temporal muscle area and cranial capacity (42 male Eskimo crania: coefficient of correlation 0.50 ± 0.08). This of course merely shows that larger crania have larger temporal muscles and bigger capacities. But if capacities diminish with increasing temporal muscle area, there ought to be no such positive correlation. Again it might be expected that the more dolichocephalic the skull the larger would be its temporal muscle areas, if the temporal muscles have the effect of restricting the lateral growth of the skull vault and causing by way of compensation an increased length. But in Eskimo crania there is no significant correlation between length-breadth index and temporal muscle area.

There is, in short, no valid evidence of any sort in favor of the supposition that increased size of the masticatory muscles either diminishes the cubical contents of the skull or alters its form.

It is well known to craniologists that the Western Eskimo show brachycephalic tendencies and that as one moves farther east in the Eskimo area the length-breadth indices decrease, culminating in the extreme dolichocephaly of the Labrador and Greenland Eskimo. I know of no dietetic influences which can account for this regional variation in head-form. Presumably the Western, Central, and Eastern Eskimo live on substantially the same diets and make similar demands upon their masticatory apparatus. It seems to me that the explanation for this difference in head-form probably lies in the fact that the Western Eskimo have intermixed to some extent with brachycephalic Indians or brachycephalic Mongoloids, whereas in the east of the area no such intermixture has taken place.

I am still inclined to regard the sagittal thickening of the skull vault, the thickening of the tympanic plate, and the palatine torus as responses to masticatory hypertrophy. But I have been somewhat shaken in my faith in the functional origin of the so-called mandibular torus by my recent study of the extensive skeletal collection from the Pecos, New Mexico, ruined pueblo. Mandibular tori are found in a high percentage of the Pecos crania, although these people must have lived principally upon maize and show only moderate development of the masticatory apparatus. What puzzles me most about the Pecos mandibular tori is that these thickenings on the lingual faces of the horizontal rami are usually in the premolar and canine region, rather than back in the molar region. If they are attributable to the lateral and rotary movements of the mandible in the vigorous mastication of tough food, it seems to me that they ought to be most prominent in the molar region where the crushing and grinding, for the most part, takes place. Of course, if it were possible to prove that these Indians made some extraordinary use of their anterior teeth such as biting off quids from plugs of hard and tough chewing tobacco, one might in that way account for the bony reinforcement of that part of the mandible, but I have not been able to find any record of such habits. As a matter of fact the exostoses on the mandible take the form usually of localized lumps rather than symmetrical ridges as in the Eskimo, and look as if they may be of pathological origin.

Nevertheless and notwithstanding, I cling to the belief that the form of the Eskimo face and jaws has been moulded to some extent by his strongly developed masticatory apparatus, although I confess that I do not know upon what meat this our Eskimo has fed, that his jaws have grown so great.

Prognathism, or jaw protrusion, is of course a relic of our anthropoidal and lower primate origin. In modern types of man this prognathism is notoriously present among many of the Negroes and Australians and to a lesser extent in most other colored races. I seriously doubt that there is any functional reason for the retention of this primatoid prominence of the jaws either in Negroes or in Australians. I am not acquainted with any use of the teeth among either of these stocks which would require or even favor the survival of this reminiscence of the snout. It seems highly improbable that diet has anything at all to do with human prognathism. The latter seems to be a recessive feature, transmitted in some stocks merely by the inertia of heredity and tending to disappear when its possessors cross with orthognathous types. Even if we admit that the Eskimo is progressive as to masticatory apparatus through function, it is none the less apparent that hypertrophy has not resulted in prognathism but rather in a widening of the jaws.

Thomson's experiment on cranial form

Many years ago Professor Arthur Thomson in a classic article on head form endeavored to show that prognathous jaws require for their operation a greater expenditure of energy by the chewing muscles, and especially more powerful temporal musculature than do less projecting jaws.* He unquestionably established this point. He also showed that in general prognathous jaws were more likely to be associated with dolichocephalic skulls than with brachycephalic skulls. Proceeding further, he devised an ingenious experiment for demonstrating the effect of temporal muscle pressure upon the sides of the skull vault and upon the shape of the brain-case as a whole. He sawed off the skull vault and affixed to the interior of the skull base a rubber bladder. He then inflated this bladder slowly, showing that, as more and more air was pumped into the bladder, its form changed from an egg shape to a shape approaching the sphere. To the sides of this inflated bladder in the regions corresponding to the areas of the temporal muscle attachments he affixed cords, fan-shaped and with their ends braided together to represent the tendons of the temporal muscles. He then showed that attaching weights to these "tendons" had the effect of narrowing the breadth of the inflated bladder and increasing

* Thomson, Arthur. "A consideration of some of the more important factors concerned in the production of man's cranial form," in *Journ. Roy. Anthrop. Inst.*, Vol. XXXIII, p. 135, London, 1902.

its length. This experiment seemed to indicate that the increased work done by the temporal muscles acting upon protruding jaws tended to repress the lateral growth of the skull and by way of compensation to increase the length.

I think, however, that such a conclusion need not be drawn. Dolichocephaly and brachycephaly are probably conditioned to a far greater extent by the shape of the relatively rigid skull base and by the size of the brain set upon that base as demonstrated by the earlier part of Professor Thomson's own experiment. If the skull base is short and broad the skull vault will be brachycephalic and the more capacious the cranial cavity the relatively greater the breadth will be. If the skull base is long and narrow the skull vault will be dolichocephalic, but the breadth will tend to increase more than the length if the cubical contents are increased. I do not believe that the pressure exerted by the temporal muscles ordinarily affects the shape of the skull vault; firstly, because the definitive head-form is attained in early infancy before the temporal muscles come into active use in masticatory movements; secondly, because the skull vault is not as plastic as an air-filled rubber bladder; thirdly, because the forces making for growth of the brain during childhood are constant, whereas masticatory pressure upon the side walls is intermittent; finally, because it seems to me no more probable that chewing narrows the head than that the active use of the muscles of the arms and back flattens and constricts the chest. Muscular use and muscular pressure do not obstruct growth; they stimulate it. In masticating food the force is always directed towards the median line, at any rate in the normal rotary movements of mastication. But this inward pressure does not reduce palatal width; on the contrary it stimulates the transverse development of the arch. Therefore it would seem that any effect of the temporal muscles upon the transverse growth of the cranial vault might well be stimulative rather than inhibitive. But I do not believe that such external forces, exerted in whatever direction, are of any great importance in comparison with the internal growth forces which shape the brain. Incidentally it may be recalled that Ivanovsky investigated the head form of Russians before, during, and after a famine and that he found that when the people were suffering from a shortage of food, and had presumably, little chewing to do, they became for the most part more dolichocephalic, since their head breadths decreased more than their head lengths.*

* Ivanovsky, Alexis. Physical Modifications of the Population of Russia under Famine. *Am. Journ. Phys. Anthrop.*, Vol. VI, No. 4, pp. 331–353, 1923.

Some one has suggested that the thick, everted, and evidently specialized lips of the Negro are to be connected with the protracted period during which Negro mothers nurse their children, but I am not aware that suckling is continued longer among Negroes than among other thinner lipped peoples living under primitive conditions.

Steatopygia

Steatopygia, the accumulation of fat on the buttocks so characteristic of the present Hottentot and Bushmen women and, apparently, of certain of the Late Palaeolithic peoples, has been regarded as an adaptive character permitting the accumulation of a reserve supply of fat to be drawn upon in lean times. Formerly I accepted this theory and even went so far as to call attention to the aestivating habits of the mouse lemur, which sleeps through the dry season, and, apparently, subsists during this period on the fat previously accumulated at the base of the tail.* In fact I was so rash as to suggest that steatopygia "may have been associated with a hibernating habit in the ancestors of the Bushmen-Hottentots and in the Aurignacian people of Europe." Today I see no merit in such a suggestion. Steatopygia is so little developed in the males of the Bushmen and Hottentots that its presence in females appears to be the manifestation of a secondary sexual character. I do not suppose that the males of these stocks are so ungallant as to take their meals regularly, leaving their wives to subsist on their own gluteal fat.

Professor Myres on the Mongol

Professor J. L. Myres, in his brilliant chapters on primitive man in the Cambridge Ancient History, commits himself to some extreme views concerning the relationship of the principal types of man to their supposed environments. For example he seems to regard the Mongol as some sort of mimetic parasite upon the horse.

> . . . It is indeed to an intimate parasitic connexion with such an animal "host," in some such circumstances, that we have probably to ascribe the highly specialized type of man characteristic of this region now. The yellow skin-colour of Mongoloid man gives him protective camouflage in sandy desert and dry-grass steppe; the structure of his straight wiry hair, and its rarity except on the scalp, suggest adaptation to a continental climate; while its extreme length in both sexes serves to disguise the characteristic profile of the human head and neck, and

* Hooton, E. A. Some Early Drawings of Hottentot Women, Harvard African Studies, Vol. II, Varia Africana II, pp. 90–99, 1918.

approximate it to that of a quadruped seen from behind. From the rather prominent jaw combined with globular brain-case may be inferred long habituation to some food which minimized the pull of the jaw muscles upon the side-walls of the skull; and the only food which fulfills this condition is milk and its products, on which nomad Tartars still live almost exclusively; the absence of face-hair, the short concave nose with spread nostrils, the peculiar infantile lips, the wide flat face and obliquely set eyes, are adaptations we should expect if for ages this milk was absorbed direct from the udder; and the short legs of some Mongoloids, and poor development of the calf muscles in all, suggest that, like Tartar infants nowadays, the parasitic proto-Mongol sat tight upon his host between meals, and shared its wanderings.*

Professor Myres even hints that some of the psychological characteristics of the Mongol may have been derived from his mount.

"Almost inhuman in his normal apathy, the Mongol can display almost equine savagery when provoked by panic or ill usage."† Detailed comment upon these passages seems superfluous. Professor Myres is one of our foremost interpreters of the beginnings of human history, a notable anthropo-geographer and a skilled archaeologist. I suspect him, in this instance, of having a little fun at the expense of his readers. At least one of them is unable to take these suggestions seriously.

Some of the most striking differences which distinguish the principal varieties of man are generally attributed, directly or indirectly, to the effects of climate upon the organism. Among these distinguishing features are pigmentation, hair form, and nose form. Since all of these are heritable, or owe their individual and group variations mainly to inheritance, one must suppose that they are adaptations hereditarily transmitted, or that they are the result of mutations which have come about in some way through environmental stimuli, or that they are chance variations perpetuated and accentuated by natural selection.

Skin Color and Climate

The case for the environmental causation of skin color is especially strong and convincing. Most of us, when we expose our bodies to the light of the sun, acquire a coat of tan. This is a deposition of melanotic pigment, mainly in the lowest layer of the epidermis, which serves as a protection against the ultra-violet rays of the sun. Those of

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* Myres, J. L. The Cambridge Ancient History, Vol. I, "Primitive Man in Geological Time," Chap. I, p. 22, 2nd. ed. New York, 1924.

† Myres, *op. cit.*, p. 23.

us who are unable to acquire such a coat of tan suffer severely from sun-burn. This protective pigmentation disappears wholly or partially when the body surface is no longer exposed to sunlight, but those who live in sunny or tropical climes, or who are constantly out-of doors, retain that pigmentation for an indefinite period. The transition to the sun-burnt Ethiopian, who has an almost black skin color which is permanent, seems easy enough to explain. He lives in a clime where the sun's rays are much more constant and powerful; he exposes all of his body to those rays; therefore his ancestors have become more and more heavily pigmented and have succeeded in transmitting that pigmentation to their offspring, so that their tissues will be the more effectively protected from injurious rays and that they may escape the slow and often painful process of tanning.

It is nevertheless apparent that there is no very close correlation between the heaviness of skin pigmentation in various human stocks and the latitudes which they inhabit. In the New World, for instance, the predominantly Mongoloid Eskimo and Indian stocks, although somewhat variable in skin color, seem to have retained a yellow-brown tone whether they live near the Arctic circle, in Tierra del Fuego, or in the equatorial regions of South America. At least I am unacquainted with any evidence that American Indians living in the Tropics are, or ever have been, black. Similarly the Mongoloid stocks of the Old World inhabit various latitudes, but we do not know that their skin color varies directly with the amount of sunlight to which they are exposed. All of the black or very dark brown races are Negro or Negroid, with the possible exception of the Australians and the so-called pre-Dravidians. Negroes transported to the temperate zone of the New World and resident there for more than three centuries seem to have lost none of their pigmentation, except through mixture with whites.

As yet no satisfactory quantitative studies of skin pigmentation have been made and prevalent anthropometric methods of judging and recording skin color are so inadequate as to be practically useless. By the use of new apparatus for the spectral analysis of colors, such scientific investigation of skin pigmentation is now possible, and the whole subject waits upon the results of a basic investigation.

The assumption that blondism is a result of a process of depigmentation in certain human stocks consequent upon a protracted sojourn in the relatively sunless region around the Baltic or some other northern area, seems to me very dubious. Fair hair, blue eyes, and sparsity

of skin pigment may well have originated in one or more human groups through mutations, but what evidence is there that such mutations were in any sense climatic adaptations? The condition known as partial albinism often is scarcely distinguishable from blondism, and albinism is at least as frequent among the dark-skinned peoples of the tropics as among the lightly pigmented races. Among the so-called "White Indians" of the San Blas tribe of Panama, are individuals who in hair color, skin color, and eye color are almost Nordic. It is abundantly evident that this tropical region has been the center of production of depigmented individuals of purely Indian blood for at least 250 years. Indeed the white Indians form approximately 0.7 per cent of the whole population in spite of the fact that white Indian men are forbidden marriage.* Albinism in this area is of genetic origin and is not progressive. It is probably due to a mutation, but it certainly has no value as an adaptation to climatic conditions. Partial albinism may be pathological, but the white Indians of Panama are not stigmatized by associated evidences of constitutional inferiority. Whether or not blondism originated from a mutation in a normally pigmented stock, it is obvious that the case of the San Blas "partial albinoes," puts the burden of proof on those who argue for an adaptational and environmental origin of the white-skinned races.

Nasal Breadth and Temperature

One of the most popular theories of man's environmental adaptation is that which relates the breadth of the nasal aperture to the temperature and humidity of air breathed, especially the former. When cold air is breathed through the nose it is warmed by passing over the highly vascular mucous membranes which line the respiratory channels. If the air is too dry the secretions of these membranes moisten it. On the other hand, the larger the amount of moist warm air which is taken into the lungs, the greater the physiological advantage. Consequently the nose serves the function of a window with a radiator behind it. In cold weather we open the window and admit the air through a narrow slit. This air passing over the radiating surface is warmed. But we do not open the window too widely; otherwise the air will not be sufficiently heated by the radiator. Thus men living in cold dry climates have narrow nasal apertures. But in the summer

* Reginald G. Harris. The San Blas Indians. *Am. Journ. Phys. Anthrop.*, Vol. IX, No. 1, p. 29, 1926.

time we turn off the radiator and throw the window wide open. So men living in the tropics have wide noses.

Professor Arthur Thomson and Mr. L. H. Dudley Buxton have developed this thesis with great ingenuity and plausibility, and have shown that there is a high and positive inter-group correlation between mean annual temperature and average nasal index, both in the living and in crania.* By means of regression formulae based upon these coefficients of correlation they have calculated mean nasal indices for a large number of groups and have compared them with actually observed means. In many cases the agreement is close; in many others there are considerable discrepancies. They explain these latter by assuming that the particular groups of mankind whose mean nasal indices deviate strongly from the calculated or predicted values have not dwelt long enough in their present habitats to have acquired the requisite nasal adaptations. Of course it is rather difficult to meet this argument in the absence of any definite knowledge as to the length of time required for the supposed adaptation and as to the period most peoples have dwelt in a stated region. According to the tables of Thomson and Buxton the Frisians have an actual mean cranial index of 40.3 whereas their calculated index is 48.3, and the Spanish have a mean index of 42 but ought to have 47.9. One supposes that the Spanish and the Frisians have been in their present habitats for a considerable length of time. Similarly the Bushmen crania tabulated (8 in number) have a mean nasal index of 60.2, whereas the prediction is 49.6, and the Hottentot crania yield an average of 57.3, although 49.6 is to be expected. But archaeology seems to indicate that the Bushman occupation of South Africa goes back a number of thousands of years.

Among the living peoples we notice the Armenians with an actual mean nasal index of 59.5 and a calculated mean index of 70.8. But surely the Armenians are no recent immigrants into the area they now inhabit. The 58 Indians of Arizona and New Mexico listed have an observed mean nasal index of 78.3, but a predicted index of 65.5. And they are no new settlers.

Nevertheless in a majority of instances the application of the reconstruction formulae of these authors seem to give moderately good results. But the exceptions scarcely prove the rule. The nasal

* Arthur Thomson and L. H. Dudley Buxton. Man's Nasal Index in Relation to certain Climatic Conditions. *Journal Royal Anthropological Institute*, Vol. LIII, pp. 92–122, London 1923.

index is the relation of the breadth of the nose to its height. If we take nasal breadth by itself we find that Eskimo (Montandon) and Guanches of the Canary Islands (v.Behr) both have average nasal breadths of 23.7 mm.; that Australians (Basedow) have a mean breadth of 27.1 and Buriats (Reicher) 27.3, while the Ainu (Koganei) and the New Irelanders (Ried) are tied at 25.6. and have identical nasal heights and indices. These are a few instances of peoples racially diverse who live in radically different climates but have substantially the same mean breadths of the nasal aperture.

On the whole I seriously doubt the validity of the argument in favor of climatic adaptation of nasal form, although I am quite prepared to admit that a narrow nasal aperture may have its advantages in a cold climate and wide open nostrils may be a benefit to the dweller in the tropics. Natural selection may operate in favor of leptorrhiny in Arctic regions and of chamaerrhiny near the equator.

I suppose that a hairy or furry covering would be of advantage to an animal dwelling in an extremely cold climate and of course we have the examples of the mammoth and the woolly rhinoceros. One might expect the Eskimo, if he is so highly adapted, to have acquired an abundant growth of body hair, but he has not such a covering, and the Australian ought to have got rid of his body hair, but he still has it.

Conclusions

In this paper I have dragged out from the evolutionary closet certain family skeletons the existence of which is often ignored. Perhaps this exposure is merely the morbid act of the exhibitionist, but I do not so regard it. My hope is that some apparently disreputable features of these skeletons will be rendered respectable by harmonizers of fact and theory more skilful than I.

The theory which evolves the human brain from that of a lowly preprimate is not open to question. The causes of such a prodigious development remain, however, obscure. Climbing a tree, sitting up on one's haunches, hand feeding and the free use of the prehensile fore-limbs—these, we are asked to believe, were the bodily habits which created anthropoid intelligence, and the human status was achieved merely by climbing down the tree and rearing up on the hind legs. It may be hard to exaggerate the effects upon the brain of these changing environments and successive bodily adaptations, but I believe they have been exaggerated.

Similarly to refer all of the hereditary physical features which distinguish present races of man to environmental factors is a specious simplification of evolutionary processes inacceptable to any but the most ingenuous mind. Of course, the acceptance of such a functional theory seems to make the biological world safe for democracy and therefore creates general satisfaction. We are unfortunately prone to identify ourselves with our biological theories. If we have a future but not a past, we are likely to be functionalists, "self-made men". If we have a past but no future we stress heredity. If we can neither glory in the past nor hope for the future we seek in environmentalism the universal alibi.

That man owes his godlike intellect to some unique creative endowment is a view displeasing to the dispassionate scientific eye; that it was foisted upon him through a fortuitous concatenation of environmental accidents is incredible. One would like to think of human evolution as a process of achievement through age-long striving of animals gifted with a divine spark of initiative, fanned into a flame by favoring winds and by a sort of spontaneous combustion. We are beginning to know something of the stages whereby man achieved his present status, but as yet, it seems to me, we are quite unable to explain the causes of human evolution.

GENEALOGICAL METHODS AS THE BASIS OF RESEARCH IN HUMAN HEREDITY

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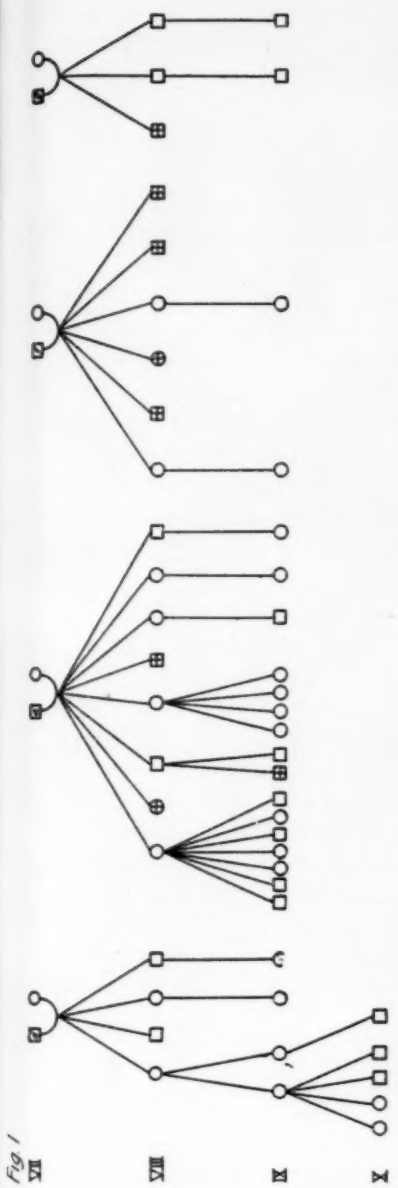
Anthropology and the doctrine of heredity are today two inseparable notions. Both sciences, however, in order to accomplish a truly beneficial work, require a third science, that of genealogy. *Anthropology without genealogy is at the present day unthinkable.* Anthropologists have occupied themselves long enough in the wholesale examination of more or less separate individuals. *A nation is composed not of separate individuals but of families, clans and blood relations.*

If we wish to determine the tendencies or diseases that are prejudicial to a nation we must try to determine the families that are infected with these tendencies or diseases. The greatest wealth of a nation consists in its free, healthy increase of population. *The principal and most weighty problem of anthropology is then family anthropology.* The genealogist treats of the doctrine of hereditary transmission just as the anthropologist does. Genealogy is therefore the given point of contact where the two sciences of anthropology and genetics, otherwise so different, meet, and can and must help one another. Genealogy must intervene and place the collected matter before the student of hereditary transmission for disposal. It is no longer an end in itself or a mere frivolity; on the contrary, it has the task of supporting and helping the researches of the student of heredity in the interest of the whole race. The "hunt for ancestors" is of the highest eugenic significance today. I shall now discuss in brief the methods of genealogical investigation and the statement of their result.

TABLES OF DESCENDANTS

In genealogical investigations we must start with the consideration that two people always stand in the mutual relationship of parent and



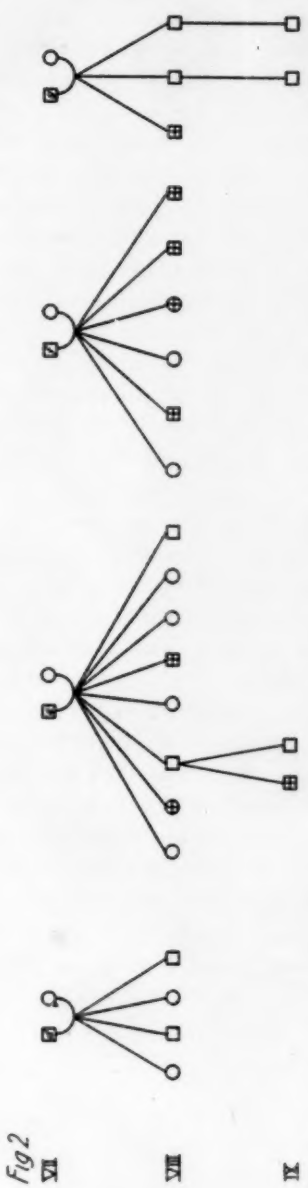


FIG. 1. PORTION OF A TABLE OF DESCENT IN THEORETICAL PURITY, SHOWING DESCENDANTS IN BOTH MALE AND FEMALE LINES

□ = men; ○ = women.

FIG. 2. PORTION OF A GENEALOGICAL TABLE OF THE SAME FAMILY AS IN FIG. 1, SHOWING DESCENDANTS IN MALE LINE ONLY

offspring. In other words in this mutual relationship we find an *active* and a *passive* side. Let us first consider the *active* side. Here we must proceed from the parent or begetter and try to determine all the descendants of his proper line which he has begotten mediately or immediately. In this manner we obtain a list of descendants or a table *in theoretical purity*. This form of statement is in any case very rare, as these investigations take up much time and are fairly expensive. The majority of students satisfy themselves with arranging the list or table in the male line, and thus consider only the direct line. This is a circumstance which is disadvantageous for the student of hereditary transmission, for the average heredity of all women, which amounts to 50 per cent, is lost. But not only is the women's heredity lost but also that of all their descendants.

It may therefore be recommended to draw up these lists or tables as far as possible in theoretical purity. In order to have a better survey of the collected results it is best to arrange them in tables. Figure 1 shows such an arrangement of a table of descent in its theoretical purity. Figure 2 shows a genealogical table, the arrangement which is in the main the most used.

By the comparison of these two arrangements a further phenomenon becomes evident which genealogy denotes as the "dying out of a generation." By this the genealogist means only the dying out in the male line. Figure 1 is a part of a list of descendants. On this we still have in the ninth generation twenty-four persons, i.e. twenty-four bearers of the same average heredity. If we consider on the other hand Figure 2, which deals with the same family, bearing in mind of course the male line only, we see that the generation is represented by only four persons, of whom one died at an early age.

From the illustrations and what has been said it follows quite clearly that *there cannot in such a case be a question of a 'dying out' from a biological point of view, but there can be from a genealogical point of view*, which, however, should not be decisive for the student of hereditary transmission.

TABLES OF ANCESTORS

If we now turn to the consideration of the *passive* side, we must proceed from the *offspring* and try to find out all its ancestors. We proceed here in just the contrary manner. As we went chronologically forwards on the list of descendants, considering things historically,

we must go chronologically backwards when considering the table of ancestors or list of ascendants.

The arrangement of a table of ancestors is the simplest form of genealogical representation which one can use of those formulas given up to the present. For in each generation the number of ances-

Maria Landwehr und Geschwister.
a Landwehr, Maria Anna, * Tilsberg, 24. 5. 1892, † Tilsberg, 5. 6. 1892.
b Landwehr, Anna, * Tilsberg, 26. 7. 1893.
c Landwehr, Wilhelmine, * Tilsberg, 18. 9. 1895.
d Landwehr, Georg Philipp, * Tilsberg, 3. 5. 1899.
e Landwehr, Georg Melchior, * Tilsberg, 9. 4. 1903.
f Landwehr, Julius, * Tilsberg, 19. 4. 1906, † Tilsberg, 16. 10. 1918.
g Landwehr, Marie, * Tilsberg, 23. 5. 1909.
1. Landwehr, Leonhardt, Schmied, * Tilsberg, 13. 6. 1864, † Tilsberg, 1. 10. 1922.
2. Brox, Augusta Regina, * Tilsberg, 2. 1. 1871.
3. Landwehr, Franz Josef, Schmied, * Tilsberg, 1. 5. 1829, † Tilsberg, 7. 8. 1897.
4. Landwehr, Barbara * Tilsberg, 13. 6. 1832, † Tilsberg, 7. 1. 1908.
5. Brox, Josef Johann, Maurer, * Tilsberg, 20. 9. 1836, †
6. Mann, Maria Eva, * Tilsberg, 21. 5. 1835, † Tilsberg, 6. 12. 1916.
7. Landwehr, Franz, Schmied, * Tilsberg, 28. 11. 1799, † Tilsberg, 9. 5. 1867.
8. Leibfried, Katharina Elisabeth, * Tilsberg, 7. 4. 1801, † Tilsberg, 30. 4. 1867.
9. Landwehr, Franz Georg, Schmied, * Tilsberg, 19. 6. 1830, † Tilsberg, 20. 1. 1879.
10. Hauer, Katharina Barb., * Neckargemünd, 28. 1. 1799, † Tilsberg, 1. 11. 1878.
11. Brox, Johannes, Maurer, * Tilsberg, 2. 1. 1806, † Tilsberg, 9. 10. 1894.
12. Weibel, Marie Elis., * Tilsberg, 23. 2. 1811, † Tilsberg, 20. 3. 1898.
13. Mann, Georg Valent., * Tilsberg, 27. 2. 1795, † Tilsberg, 28. 7. 1856.
14. Haas, Maria Anna, * Tilsberg, 1799, † Tilsberg, 18. 11. 1874.
× Heidelberg, 31. 10. 1891.
× Tilsberg, 27. 11. 1852.
× Tilsberg, 3. 10. 1822.
× Tilsberg, 13. 11. 1825.
× Tilsberg, 20. 1. 1831
× Tilsberg, 18. 2. 1864.
× Tilsberg, 16. 11. 1830

FIG. 3. TABLE OF ANCESTORS * = births; † = deaths; × = marriages.

tors increases twofold. Each human being has two parents, four grandparents, eight greatgrandparents and so on, or mathematically expressed, in the *n*th ancestral generation, the number of ancestors is 2^n.

Figure 3 shows a simple statement of the table of ancestors.

With regard to the numbering of the ancestors on the table it is on the whole customary to denote the propositus with 1, the father with 2, the mother with 3, and so on. This system of numbering as such is false, *for the propositus is no ancestor.* We express him separately in numbering.

From the old system of numbering the number of ancestors in a given generation stands out at once, but this is also the case in the methods suggested and applied by me. To obtain it one must add 1 to the first number appearing in the line. Now we must solve the question as to how far one ought to trace a pedigree. *The task of the student of hereditary transmission is to trace a family not vertically but in the collateral line.* It would thus suffice for a pure hereditary biological end to draw up a table of ancestors up to the greatgrandparents. For the generations which precede this the necessary biological sources are usually lacking. We have still in this eight-fold table an *average* hereditary value of 12½ per cent for each greatgrandparent. We can read the percentage of *average* heredity from the table of ancestors. The first generation has an average percentage of 50 per cent, the second of 25 per cent and the third of 12½ per cent. I emphasize expressly that only average values are here considered and also that the sex chromosome has not been taken into account.

These tables will be especially useful if the photograph of each person is put in the place belonging to him. Racial characteristics whose hereditary progress brings forth special facial characteristics, and so on, thus stand out clearly. Such an arrangement is shown in Figure 4.

It might be recommended here that the photographs be taken from a strictly scientific standpoint without being retouched, perhaps three views of each person as shown in Figure 5.

INBREEDING

Along with the discussion of the ancestor table I shall now examine a phenomenon which is especially valuable for the student of hereditary transmission; this is the so called "*loss of ancestors*" (*Ahnenverlust*). The *loss of ancestors is the phenomenon on the ancestry table which is brought about by marriage of blood relatives and allows the same people to appear two or three times on the same table.* The designation of "loss of ancestors" is unhappily chosen; for every human being has always the same number of ancestors, namely, as already stated above, two parents, four grandparents, eight greatgrandparents, etc. A "*loss*"

FIG. 4. ILLUSTRATED TABLE OF ANCESTORS (*Facing page 254.*)

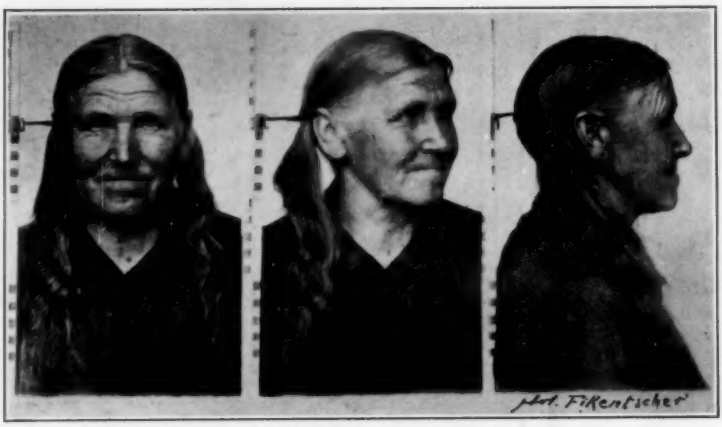


FIG. 5. THREE VIEWS OF A SUBJECT

of ancestors cannot enter into the question. Sommer recognized this phenomenon and chose for it the expression "*concentration of ancestors*"

7	8	9	-10	11	12	13	14
Maurer, Sebastian, *Mainz 16-7-1777 +Dilsberg 11-6-1818	Hallbauer, Maria, *Heidelberg 28-4-1780 +Dilsberg 23-4-1835	Mann, Franz, *Dilsberg 15-8-1783 +Dilsberg 17-12-1851	Mann, Eva, *Dilsberg 25-10-1781 +Dilsberg 25-12-1850	Nr. 9	Nr 10	Werner, Georg, *Dilsberg 14-3-1784 +Dilsberg 26-10-1834	Breuner, Margarethe, * Dilsberg 14-3-1784 + Dilsberg 7-4-1853
×Dilsberg 9-6-1801	× Dilsberg 3-4-1808					×Dilsberg 4-1-1809	
3 Maurer, Johann, * Dilsberg 23-5-1802 + Dilsberg 14-8-1864		4 Mann, Katharina, * Dilsberg 27-11-1808 + Dilsberg 15-4-1877		5 Mann, Georg, * Dilsberg 17-8-1811 + Dilsberg 28-2-1890		6 Werner, Margarethe, * Dilsberg 3-10-1809 + Dilsberg 18-11-1880	
× Dilsberg 26-6-1834				× Dilsberg 9-10-1838			
1 Maurer, Johann, Georg,	*Dilsberg 28-2-1838 +Dilsberg 22-4-1886	× Dilsberg 27-11-1862		2 Mann, Anna Maria,		* Dilsberg 28-1-1840 +Dilsberg 1-3-1920	
Maurer, Georg, *Dilsberg 28-2-1881							

FIG. 6. ANCESTRY TABLE SHOWING EQUATION OF ANCESTORS

(*Ahnenkonzentration*). But here also the designation is false, for a concentration of ancestors can no more enter into the question than a loss of ancestors. That which is concentrated is merely the *hereditary*

mass (*Erbmasse*). Scheidt proposed therefore the designation "*hereditary accumulation*" (*Erbhaüfung*), which is the best term yet suggested.

7 Brack, Christian, *Darmstadt 14-1-1819 +Darmstadt 25-5-1901	8 Ruckel, Sophie, *Darmstadt 24-12-1819 +Darmstadt 8-5-1890	9 Rind, Heinrich, *Darmstadt 8-11-1818 +Darmstadt 17-9-1897	10 Gust, Margarethe, *Giessen 18-6-1820 +Darmstadt 22-3-1894	11 Löhn, Konrad, *Nürnberg 6-3-1845 +Würzburg 24-7-1878	12 Schön, Maria, *Bamberg 17-6-1848 +Würzburg 5-5-1907	13 Nr. 3	14 Nr. 4
xDarmstadt 25-2-1841		xDarmstadt 6-7-1845		xBamberg 3-11-1867			
3 Brack, Ludwig, *Darmstadt 7-10-1843 +Darmstadt 24-12-1921		4 Rind, Anna, *Darmstadt 6-8-1851 +Darmstadt 5-7-1917		5 Löhn, Friedrich, *Würzburg 27-2-1872		6 Brack, Anna, *Darmstadt 8-5-1875	
x Darmstadt 2-10-1874				x Würzburg 23-6-1897			
Brack, Karl, *Darmstadt 30-5-1892		xWürzburg 14-5-1926		Löhn, Berta, *Würzburg 1-3-1904			
a Rosemarie *Würzburg 28-6-1926		b Karl, *Nürnberg 3-5-1927		c Hertha, *Nürnberg 25-8-1928			

FIG. 7. ANCESTRY TABLE SHOWING DISPLACEMENT OF ANCESTORS

In the ancestry table one can distinguish two types of hereditary accumulation or "inbreeding" (*Inzucht*):

Closer inbreeding or *equation of ancestors* (Ahnengleichung)

and

Broader inbreeding or *displacement of ancestors* (Ahnenverschiebung).

Equation of ancestors or stricter inbreeding comes in between first cousins.

The expression "equation of ancestors" is derived from the fact that those persons who appear twice appear in the *same ancestral degree.* An ancestry table with equation of ancestors is shown in Figure 6.

Displacement of ancestors or broader inbreeding comes in a marriage between uncle and niece or aunt and nephew. Thence comes the expression "*displacement of ancestors,*" because the ancestors who appear twice appear in *different degrees* of ancestry. In Figure 7, for example, we see the doubled ancestors once in the grandparental generation and once in the greatgrandparental. Thus a glance at the table suffices

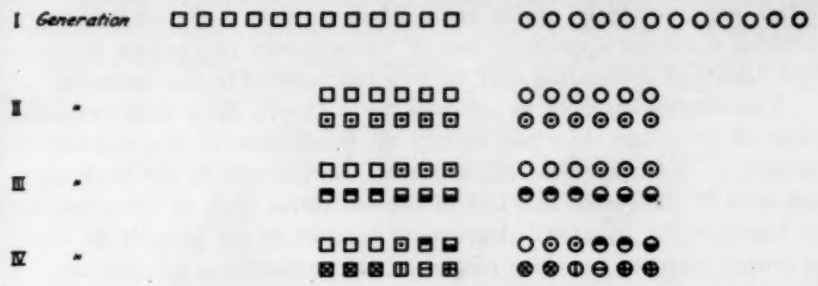


FIG. 8. THE DESCENT OF CHROMOSOMES, SHOWING UNEQUAL INHERITANCE FROM DIFFERENT ANCESTORS

to ascertain what kind of inbreeding is in question. Naturally the average hereditary values may be reckoned mathematically from these tables.

The ancestry table shows us now the building up of our own "Ego." That is, it shows all the persons (up to a certain grade) who gave parts of their inheritance to the propositus or propositi. *But what this table lacks are the brothers and sisters of all persons appearing on this ancestry table.* Even if we are able to reckon in the average value of the hereditary mass (*Erbmasse*) of the separate individuals, these figures are still too arbitrary. I shall try to make this clear by an example. Figure 8, without going further into chromosome doctrine (*Chromosomenlehre*), a knowledge of which I have taken for granted. As already known the ova and sperm meet together with half the number of chromosomes (*Chromosomenzahl*) corresponding to the species (in the case of man twenty-four), and even here I leave the sex

chromosome (*Geschlechtschromosom*) out of account. In the reducing division half of the chromosomes are expelled. But there do not always remain behind the same chromosomes *as the mixing of the parental chromosomes will always be different in the individual cells.*

To make this more easily understood the following sketch will serve. The squares denote male chromosomes and the circles female. This representation always permits of further improvement but it should suffice for the case before us. We see here that the chromosomes of the grandparents are no longer represented in altogether equal numbers; this could naturally have been true in earlier generations. In the example chosen here the paternal grandfather's side preponderates in the fourth generation. If we now change the chromosomes at will we shall find it easy to understand that brothers and sisters are frequently unlike each other, or that a certain tendency towards a disease appears in one of the propositi but not in another. The results of inbreeding may be well represented in this example.

This digression may be allowed me. It will show that even the table of ancestors does not satisfy all the desires of the student of heredity. We must proceed, as already mentioned in the beginning, not only in the direct line but in the collateral, that is, we must try to examine the collateral degrees as completely as possible in order to obtain a somewhat clear picture of the connections in question.

DATA OBTAINABLE FROM TABLES

Before I discuss the third form of genealogical representation let us next propose the question of the difference between these two tables discussed up to the present and their value for the student of heredity. In clear scientific consideration the difference between the tables lies in the fact that on the table of descent a "*thinning out*" is observable, while on the table of ancestors there is an "*accumulation*" or *concentration.* Considering the table of descent more minutely with regard to dates (which I have not mentioned in the chosen examples), we get a whole string of questions of a biological nature.

Just to mention a few we have:

1. Marriage age of the woman and number of children.
2. Marriage age of the man and number of children.
3. Occupation and number of children.
4. Age at death of the man and children.
5. Age at death of the woman and children.
6. Proportion of male births to female births.

7. Infant mortality and number of children.
8. Interval between births and infant mortality.
9. Cases of multiple births; and so on.

Further, the determination of the *causes of death* is valuable. For a single individual, it is true, it proves little or nothing, but considered in relation to a family table it gives the student of hereditary transmission a few valuable hints.

We can put aside the table of ancestors for

1. Hereditary biological research.
2. Sociological research.
3. Historico-biographical research.

TABLES OF CONSANGUINITY

Now for the representation of the third type of table. As has been mentioned already, neither the family table nor the table of ancestors suffices. Czelitzer, a Berlin oculist, who gave much attention to

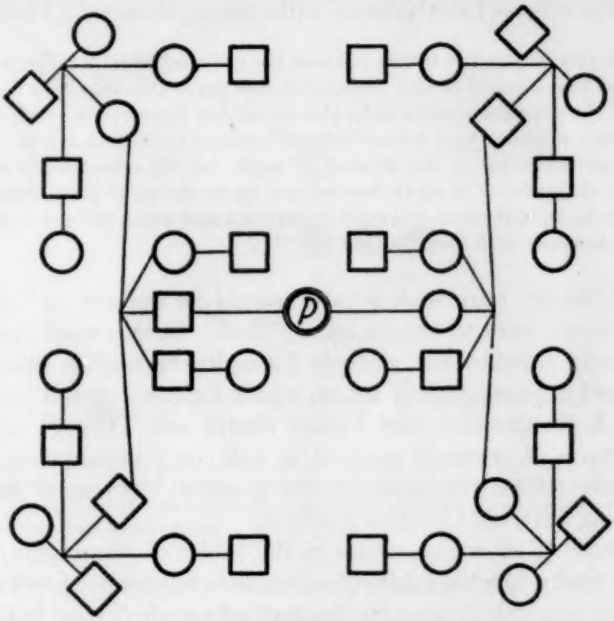


FIG. 9. TABLE OF CONSANGUINITY

the question of hereditary transmission, recognized the deficiency and combined the two tables into one. Figure 9 shows such a table, which I have changed somewhat for practical purposes and show in

the examples, Figures 10b and 11b. These tables when illustrated with the pictures of the persons under consideration are of special value. Czelitzer called this a *table of consanguinity or of kindred.* The designation Table of Consanguinity is the more comprehensible and the better. Opinions are still rather divided on the idea of kindred. According to Stradonitz the conception of kindred is:

1. Family in the narrower sense of the word—parents and children.
2. Kindred—ascendants and descendants.

Scheidt describes this more exactly; his classification is:—

1. Family—consorts and children.
2. Clan (i.e. family in the widest sense), members of the family and all brothers and sisters of the consorts plus their parents and their brothers and sisters, etc.
3. Kindred, members of the clan and of the kin plus consorts of the brothers and sisters of the forefathers and their offspring.

With this formulation Scheidt comes the nearest to the claims of Devrient, who reproaches Czelitzer with incompleteness. Devrient says

> "Evidently it does not suffice to know the parents, grandparents, brothers and sisters and first cousins of the propositus: one must also take into consideration the kin of the consorts in order to be able to cut out those taints which come to the cousins from ancestors who are not related by blood to the propositus. We should take the ancestors up to the number of eight, all the descendants of the eight ancestors, the consorts of their descendants up to the same generation and again the descendants of these to the same generation and again the entire descendants of those ancestors with their consorts."

I admit readily that such a table would be defined as "*ideal*". It will, however, have to remain but an ideal. Such a table can be made for princely families but scarcely for ordinary middle class families. I arranged a consanguinity list on which I guided myself according to the Tirol, Switzerland, the United States and Turkey; and despite *several years* of research work it is still very incomplete. We can move only within the limits of the possible. We must be satisfied if we bring together Czelitzer's tables.

In order to show the nature of the tables of consanguinity I shall contrast them with the tables of ascendants by means of two examples. In the first example, Figure 10, the married couple *N* have four children, a girl and three boys. The three boys were born blind; two of them died young and the third is in a blind institution. Now from whom came the blindness? The table of ascent throws no light on the question, but with the help of the table of consanguinity we meet with

a distinctly better result. While the table of ascent proved nothing at all, we can see on the table of consanguinity that two brothers of the mother of the person in question were blind, as well as a brother of the mother's father (Fig. 10b).

In the second example, Figure 11, the table of consanguinity treats the inheritance of congenital cataract with the same success as above.

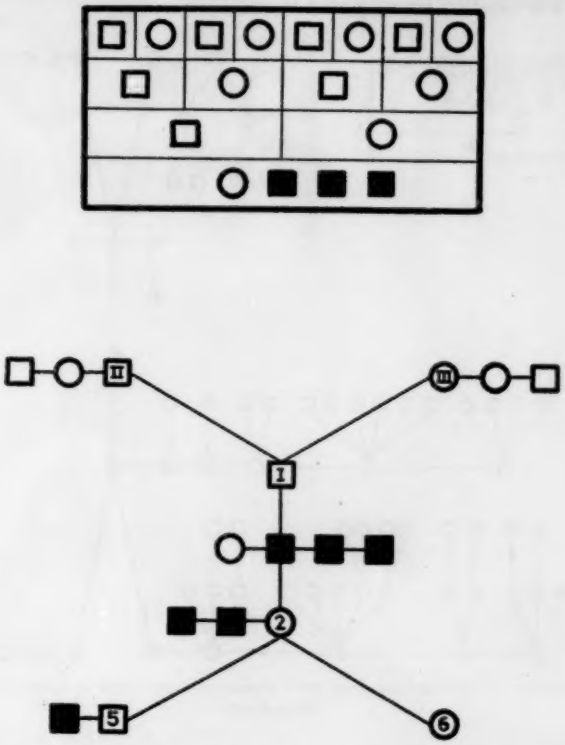


FIG. 10. TABLES OF ANCESTRY AND CONSANGUINITY FOR THE SAME FAMILY While the table of ancestry throws no light on the inheritance of the cases of congenital blindness denoted by the black squares, the table of consanguinity shows that it has occurred in previous generations.

(For the numbering of the table of consanguinity it is advisable to express the *male ancestry with Roman numerals and the female with Arabic*).

With these arguments and the quoted examples I hope to have supplied the proof that genealogical methods form the basis of research in human heredity. I hope furthermore to have supplied the proof

that in genealogical researches for hereditary biological ends it is necessary to trace the families not only in the direct line but also in the collateral. It would be a thankful task to study, as I have done for a long time, the inmates of blind and deaf and dumb institutions, etc., in order to lay down in this manner a plan of health for the rising generation; a task which is very laborious but which from a eugenic point of view should be of great value.

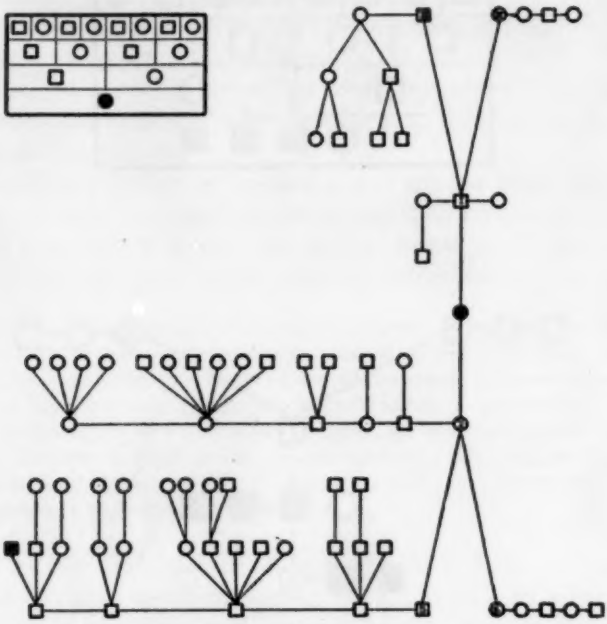


FIG. 11. TABLES OF ANCESTRY AND OF CONSANGUINITY FOR A CASE OF CONGENITAL CATARACT

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NOTES ON BLOOD PRESSURE IN OLD AGE[1]

BY H. JACKSON DAVIS, M.D., DR.P.H.

I

The objects of these notes are: (1) to record the basic biometric constants from blood pressure data on 50 white males over the age of 75 years admitted to the medical service of the Johns Hopkins Hospital, Baltimore; (2) to record the same constants for the data of Thompson and Todd[2] on 102 white males in the same age group in the Royal Hospital, Chelsea; and (3) to compare the statistical constants thus obtained with each other and with similar data in the literature.

Except for the paper by Saller[3] of Kiel, the writer knows of no attempt at a thorough statistical analysis of blood pressure data in this age group, although some primary data may be found in the papers of Bowes,[4] Wildt,[5] and Christensen.[6]

[1] From the Institute for Biological Research of the Johns Hopkins University.

[2] Thompson, R. J. C., and Todd, R. E. Old age and blood pressure problems. *Lancet, London*, 1922, ii, 503. Gives frequency distributions of systolic, diastolic and pulse pressures taken on 102 men, from 75 to over 90 years of age, at the Royal Hospital, Chelsea.

[3] Saller, K. Über die Altersveränderungen des Blutdrucks. *Ztschr. f. d. ges. exper. Med.*, 1928, lviii, 683–709. Contains a study of the systolic and diastolic blood pressures of 7382 men and 5197 women in senile and presenile age groups. There is some statistical analysis of these data. See Tables 6 and 7 for constants cited.

[4] Bowes, L. M. Blood pressure in the aged. A study of 150 cases from 65 to 95 years of age. *Journ. Lab. and Clin. Med., St. Louis*, 1917, ii, 256–259. A study of 100 women and 50 men. Source of series is not mentioned. Contains frequency distributions and mean systolic, diastolic and pulse pressures by 5-year age groups between 65 and 94 years. Data cited in Tables 6, 7, and 8.

[5] Wildt, Hermann. Über Blutdruck im Greisenalten. (Inaug. Dissert. . . . zur Erlangung der Doktorwürde. *Strassburg.*) 1912. Brochure. 12p. Also in *Zentralbl. f. Herzkrkh. u. Gefässkrkh.*, 1912, iv, 49. A study of 250 cases of both sexes, ages 50 to 96 years. Contains frequency distributions and mean systolic, diastolic and pulse pressures by 5-year groups between 60 and 89 years. Cases between 90 and 96 years comprise the last group. Data cited in Tables 6, 7 and 8.

[6] Christensen, Håkon B. Undersøgelser over det intraokulaere Truk og Blodtryk has gamle Folk. København, 1910. *Hosp. Tid.*, 5, Raekke, iii, nr. 50,

The systolic blood pressure represents the maximum cardiac energy at a given moment, together with the degree of peripheral resistance; the diastolic blood pressure represents the minimum cardiac energy at a given moment, and is a measure both of peripheral resistance and of vasomotor tone; while the pulse (differential) pressure is not a

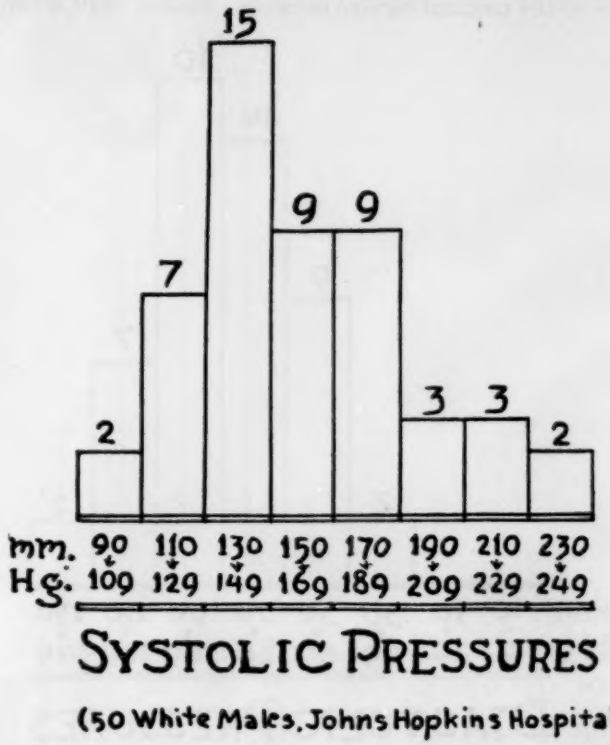

FIG. 1. HISTOGRAM OF VARIATION IN SYSTOLIC PRESSURE. JOHNS HOPKINS

direct measure like the maximal and minimal pressures, but the difference between these two variables. The systolic and diastolic pressures are conventionally measured by compression of the brachial artery by an instrument which measures the pressures in terms of the height in millimeters of a column of mercury. The two best types of instrument are the mercurial and aneroid sphygmomanometers.

(1393)–1397. Gives systolic and intraocular pressures, age, and sex for 152 patients in the Kopenhagen Home for the Aged, ages 62–91. The average systolic blood pressure for the 94 cases with normal intraocular pressure was 160–161 mm. Hg.

Two series of records were used in this study, namely:

A. From the case histories of the Johns Hopkins Hospital medical service, systolic, diastolic and pulse pressures of 50 white males over the age of 75 years were compiled. These comprise all histories, containing blood pressure data in this age, sex and race group, of admissions to the medical service between October, 1912, when readings

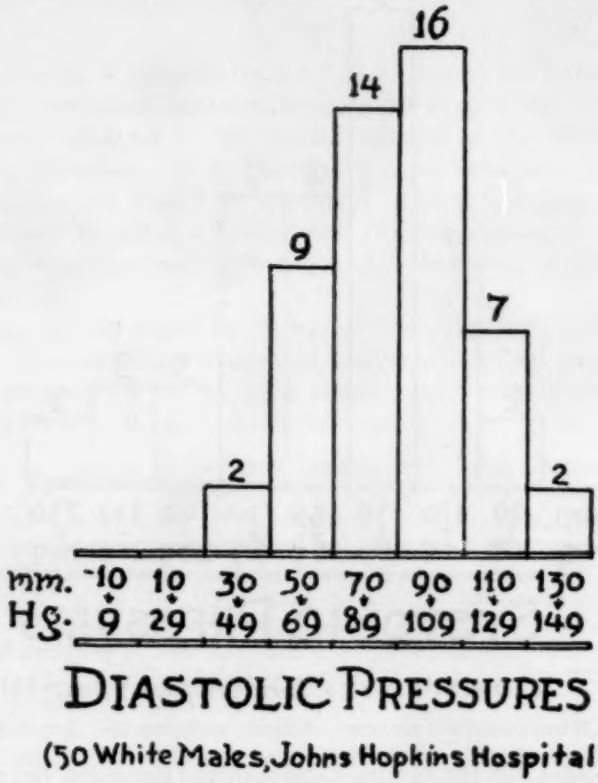

FIG. 2. HISTOGRAM OF VARIATION IN DIASTOLIC PRESSURE. JOHNS HOPKINS

were first recorded, until the middle of 1924. The original data, chronologically arranged, may be examined in the Appendix.

The frequency distributions are plotted in Figures 1–3.

B. From the Royal Hospital, Chelsea, the systolic, diastolic and pulse pressures of 102 white males over the age of 75 years, published in the paper of Thompson and Todd, were used.

The frequency distributions are plotted in Figures 4–6.

II

The statistical constants determined for the two sets of data are shown in Tables 1 and 2.

In Tables 3–5, the constants for the systolic, diastolic, and pulse pressures of the Johns Hopkins and Royal Hospital data are compared,

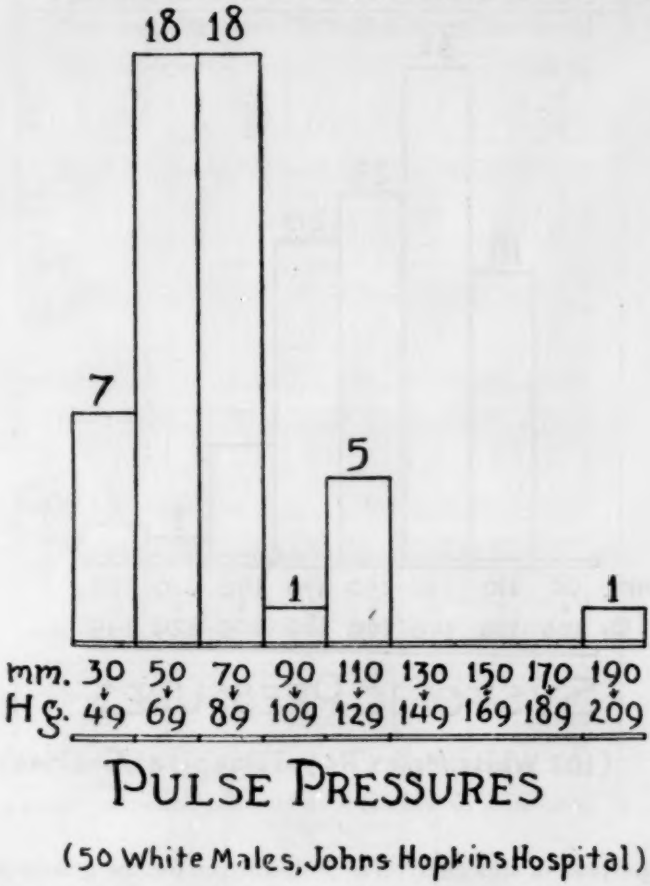

FIG. 3. HISTOGRAM OF VARIATION IN PULSE PRESSURE. JOHNS HOPKINS

the differences determined, and the statistical significance of these differences, in terms of their probable errors, duly noted.

In Tables 6–8, certain constants from the literature are tabulated, to facilitate comparison with the constants recorded in Tables 1 and 2.

It is not the province of this limited study to discuss the clinical or diagnostic significance of blood pressure readings in relation to old age. Conclusions of that nature would not be justifiable in view of the small number of cases studied, the rather highly selected nature of the groups, and the paucity of other pertinent data which are necessary to a rational evaluation.

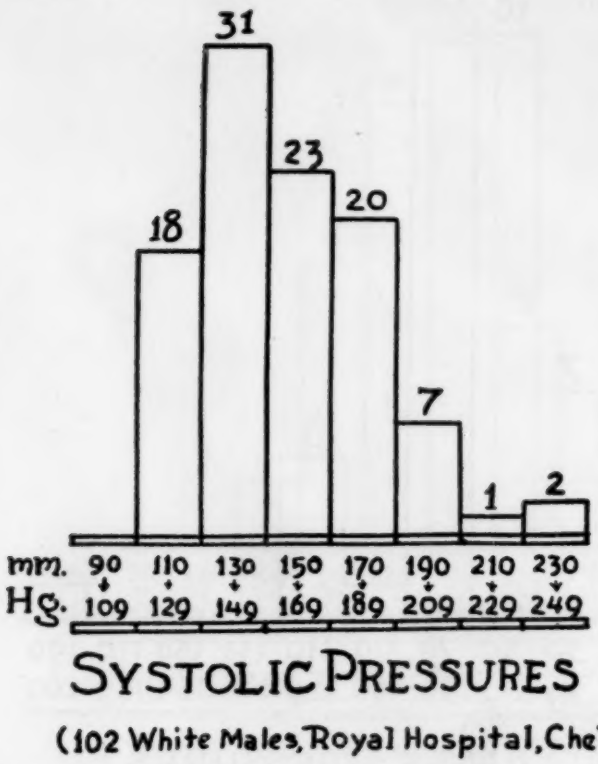

FIG. 4. HISTOGRAM OF VARIATION IN SYSTOLIC PRESSURE. CHELSEA

The purpose of this analysis is to determine for the groups studied the central, average values about which the pressures fluctuate, the scatter measures—i.e., the standard deviation in absolute terms, and the coefficient of variation, a comparative or relative measure of variation, in percentage terms.

In the interpretation of these statistical constants, the nature of the two groups studied should be kept in mind.

The 50 men admitted to the medical service of the Johns Hopkins were all, presumably, Americans. They were not selected cases from this service, but were all cases in the age, sex and race group under study, on whom systolic and diastolic blood pressures were recorded.

RESULTS: STATISTICAL CONSTANTS.

	MEAN in mm Hg	MEDIAN in mm Hg	MODE in mm Hg	STAND. DEV. mm Hg	COEF. or VAR. mm Hg
Table 1. Constants based on Johns Hopkins Blood Pressure Data on 50 Men-Age 75+					
Syst.	158.3 ± 3.2	151.7 ± 4.0	138.6	33.1 ± 2.2	20.9 ± 1.5
Diast.	88.7 ± 2.2	89.5 ± 2.7	91.1	22.7 ± 1.5	25.6 ± 1.8
Pulse	73.5 ± 2.6	69.5 ± 3.3	61.5	27.5 ± 1.9	37.4 ± 2.9
Table 2. Constants based on Royal Hospital Blood Pressure Data on 102 Men-Age 75+					
Syst.	155.2 ± 1.8	151.2 ± 2.2	143.3	26.5 ± 1.3	17.1 ± 0.8
Diast.	78.1 ± 1.2	79.1 ± 1.4	81.1	17.3 ± 0.8	22.1 ± 1.1
Pulse	77.7 ± 1.6	74.5 ± 2.0	68.0	23.9 ± 1.1	30.8 ± 1.6
Table 3. Comparison of Constants on Systolic Pressure Data from Tables 1 & 2					
J.H	158.3 ± 3.2	151.7 ± 4.0	138.6	33.1 ± 2.2	20.9 ± 1.5
R.H	155.2 ± 1.8	151.2 ± 2.2	143.3	26.5 ± 1.3	17.1 ± 0.8
Diff.	3.1 ± 3.6	0.5 ± 4.5	-4.7	6.6 ± 2.6	3.8 ± 1.7
Signif.	Diff < P.E. diff.	Diff < P.E. diff.	—	Diff = 2.5 × P.E. diff.	Diff = 2.3 × P.E. diff.
Table 4. Comparison of Constants on Diastolic Pressure Data from Tables 1 & 2					
J.H	88.7 ± 2.2	89.5 ± 2.7	91.1	22.7 ± 1.5	25.6 ± 1.8
R.H	78.1 ± 1.2	79.1 ± 1.4	81.1	17.3 ± 0.8	22.1 ± 1.1
Diff.	10.6 ± 2.5	10.4 ± 3.1	10.0	5.4 ± 1.7	3.5 ± 2.1
Signif.	Diff = 4.3 × P.E. diff.	Diff = 3.4 × P.E. diff.	—	Diff = 3.1 × P.E. diff.	Diff = 1.7 × P.E. diff.
Table 5. Comparison of Constants on Pulse Pressure Data from Tables 1 & 2					
J.H	73.5 ± 2.6	69.5 ± 3.3	61.5	27.5 ± 1.9	37.4 ± 2.9
R.H	77.7 ± 1.6	74.5 ± 2.0	68.0	23.9 ± 1.1	30.8 ± 1.6
Diff.	-4.2 ± 3.1	-5.0 ± 3.8	-6.5	3.6 ± 2.2	6.6 ± 3.2
Signif.	Diff = 1.3 × P.E. diff.	Diff = 1.3 × P.E. diff.	—	Diff = 1.7 × P.E. diff.	Diff = 2.1 × P.E. diff.

Table 6. Syst. Pr. Constants from Literature					Table 7. Diast. Pr. Constants from Literature			
MEAN	STAN. DEV.	COEF. VAR.	FREQUENCY	SOURCE	MEAN	STAN. DEV.	COEF. VAR.	FREQUENCY
158.6			29 of 75-94	Bowes (3)	86.0			29 of 75-94
144.5			50 of 75-89	Wildt (4)	70.0			50 of 75-89
156 ± 2.6	25.1	18.5	83 of 68-89	Sailer (2)	78 ± 1.7	25.1	21.5	83 of 68-89

Table 8. Pulse Pr. Consts. from Lit.		
MEAN	FREQUENCY	SOURCE
76.5	29 of 75-94	Bowes (3)
74.5	50 of 75-89	Wildt (4)

In each case, the blood pressure on admission was used, to eliminate the influence of treatment. The observations were, for the most part, made with a mercury sphygmomanometer.

The data of Thompson and Todd comprise "systematic readings of 102 in-pensioners from 75 to 92 years of age" in the Royal Hospital,

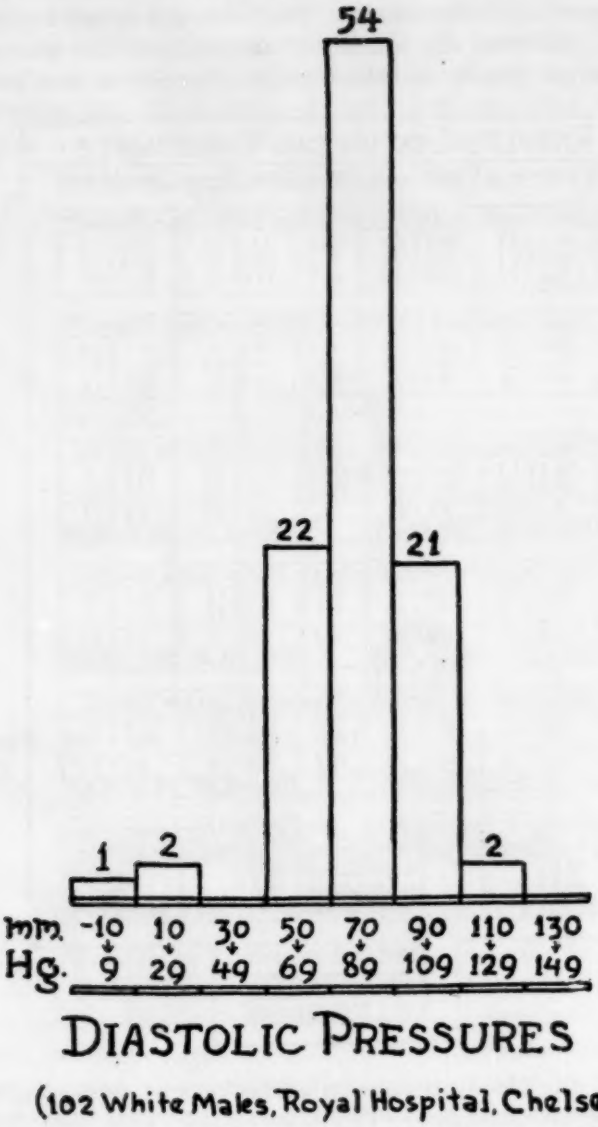

FIG. 5. HISTOGRAM OF VARIATION IN DIASTOLIC PRESSURE. CHELSEA

Chelsea. The Tycos (aneroid) sphygmomanometer was used in their study.

III

There is no significant difference between the average values for systolic pressure in the Johns Hopkins and Royal Hospital data. The

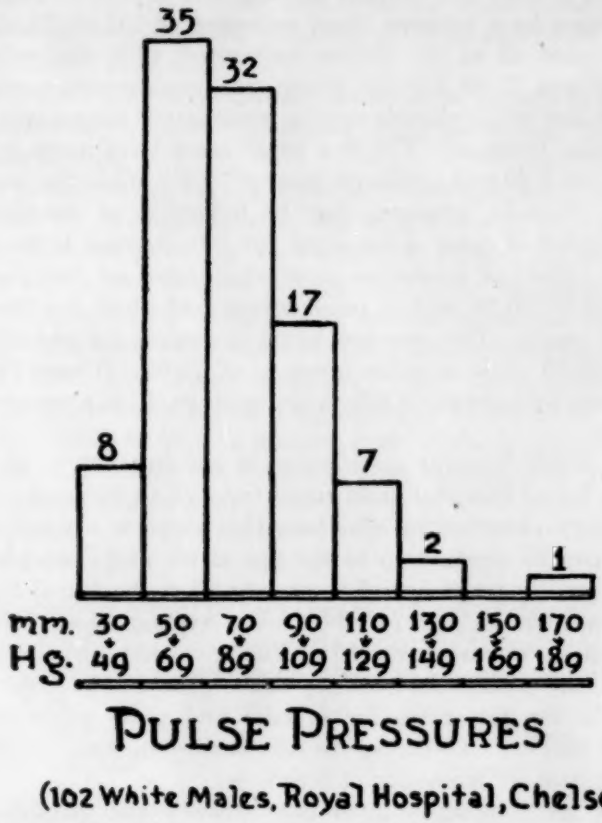

Fig. 6. Histogram of Variation in Pulse Pressure. Chelsea

differences between the variability measures, while somewhat larger, are not significant (See Table 3).

The differences between the average values for diastolic pressure are more than three times their probable errors, and are therefore to be regarded as probably statistically significant (See Table 4).

It has been axiomatic that the diastolic pressure is an index of the cardiac load, and that this load is increased by abnormal conditions in the heart and kidneys. Two observations are relevant to the greater values for the Johns Hopkins data.

(1) Forty-two per cent, or 21 of the 50 cases have had a clinical diagnosis of cardiac, renal, or cardio-renal disorder (see Appendix), distributed as follows: 13 cardiac; 5 renal; and 3 cardio-renal. The 13 cardiac cases have average blood pressures of 152.38/79.92, with a pulse pressure of 81.23. When contrasted with the values of 158.30/88.70 and 73.50 for the group, the compensatory response of the mechanism to the chronic cardiac insufficiency is apparent in the increased pulse pressure. The five renal cases have average blood pressures of 168.4/94 and a pulse pressure of 74.4. While the increased systolic and diastolic pressures may be indicative of the increased load, the number of cases is too small for this increase to be of any significance. Perhaps somewhat more significant are the increased values of 169.00/99.33 with a pulse pressure of 69.67 for the three cardio-renal cases. The average blood pressures for the 21 cases are 163.26/91.06 with a pulse pressure of 75.09. These 21 cases obviously have a considerable rôle in the increase of the average values for the group.

(2) The Johns Hopkins cases range in age from 75 to 83 years, whereas the Royal Hospital cases range from 75 to 92 years. It has been frequently observed by clinicians that there is a tendency for blood pressures to increase up to the age of 80, and then gradually drop. The greater frequency of cases over 80 in the Royal Hospital group may account in part for their lower average blood pressures.

The influence of cases over 80 on the frequency distributions of both groups may explain the tendency to skew toward the lower values observable in the histogram for systolic and pulse pressures (See Figs. 1, 3, 4, and 6.). Of course the samples are too small to give any value to a statistical expression of skewness.

There is no significant difference between the coefficients of variation of the diastolic pressures. The difference between their standard deviations is more than three times its probable error, and hence probably statistically significant. This difference may be due to the influence of the 21 known abnormal cases in the Johns Hopkins group under consideration.

There is no significant difference between either the average values or the scatter measures for the pulse pressure data (See Table 5).

The constants collected from the literature in Table 6, 7, and 8, are presented for reference and comparison with the constants in Tables 1 and 2. It is inadvisable to draw any conclusions from this comparison because of the scanty data available relative to the frequency distributions on which these constants were based.

The coefficients of variation, or relative scatter measures, for these data range from 37.41 and 34.92 for pulse pressure, through 25.56 and 22.1 for diastolic pressure, to 20.86 and 17.06 for systolic pressure (See Tables 1 and 2).

For the purposes of comparison it may be of interest to the clinician to compare these scatter measures with other familiar biometric constants in man.

The coefficients of variation for pulse pressure in this study, 37.41 and 34.92, are of approximately the same order as the constants for the weight of the spleen in the healthy, 38.21, and the weight of the heart in the general hospital population, 32.39. Similarly, the coefficients of variation for diastolic and systolic pressures, ranging from 25.56 to 17.06, are of about the same order as the constants for keenness of sight, 28.68, and breathing capacity, 16.6. It is noteworthy that all of these constants are of a significantly higher order than those for body weight, 10.37, and stature, 3.99, in the English. All of the constants cited above are from the table compiled by Pearl[7] in his monograph on brain weight.

IV

In conclusion it may be said that the object of this study has been to put on record the chief biometric constants of individual variation in blood pressure among persons falling in age near the upper end of the life span. Two different series of data (one English and one American) give nearly the same mean and median values for systolic pressures, 155–158. The mean diastolic pressure is lower in the English series (79 against 89 in the American series). Other biometric constants are discussed in the text.

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[7] Pearl, R. Biometrical studies on man. I. Variation and correlation in brain-weight. *Biometrika, Cambridge*, 1905, iv, 13–104. In table VII, page 32, coefficients of variation for man are presented in order of magnitude.

APPENDIX

Original Blood Pressure Data (Systolic, Diastolic, and Pulse Pressures in mm. Hg.), Age, and Diagnoses on Fifty White Males, Ages 75–83 Years admitted to the Medical Service of the Johns Hopkins Hospital between 1912 and 1924

CASE	AGE	SYST.	DIAST.	PULSE	DIAGNOSES
1	83	215	140	75	Arterio-sclerosis
2	80	172	122	50	Tubero. left wrist
3	75	145	92	53	Bronchitis, Acute
CR 4	76	180	120	60	Art. scl., Chr. Nephr., Myocard. Insuff., Auric. Fibrillation.
5	81	170	130	40	Art. scl., Hypertension, Emphysema
R 6	75	192	120	72	Art. scl., Chr. Nephr., Hypertension, Emphys.
7	76	208	96	112	Art. scl., Myocard. Insuff., Hypertroph. Prost.
8	76	145	95	50	Cerebral Thrombosis
9	75	120	75	45	Art. scl., Carcinoma Stomach, Secondary Anemia, Pyloric Obstruction.
10	82	172	122	50	Art. scl., Hypert., Carc. Pancreas.
R11	75	160	80	80	Art. scl., Senility, Renal Insuff.
12	79	130	60	70	Arthritis Spine, Peridental Abscess
13	80	140	70	70	Chr. Peritonitis, Perihepatitis, Perisplenitis.
R14	76	230	120	110	Art. scl., Hypertension, Oral Sepsis, Nephropathy
15	75	160	100	60	Art. scl., Psychoneurosis
16	75	142	60	82	Chr. Arthritis, Chr. Inflam. Lumbar Spine.
17	75	130	70	60	Art. scl., Carc. Prost., Carc. Intest., Secondary Anemia.
18	76	180	94	86	Achylia Gastrica
C 19	76	200	90	110	Art. scl., Hypertens., Myocard. Insuff., Auric. Fibrillation.
C 20	76	140	90	50	Art. Scl., Chr. Myocard., Myocard. Insuff.
21	81	120	55	65	Secondary Anem., Oral Sepsis, Hypertrophied Prost., Undernutrition.
C 22	75	210	95	115	Art. scl., Hypertens., Cardiac Hypertrophy, Myocard. Insuff.
23	75	160	90	70	Art. scl., Hypertens., Card. Hypertrophy
C 24	76	124	80	44	Art. scl., Myocard. and Mitral Insuff., ?Carc. Stom., Secondary Anemia.
25	76	162	90	72	Art. scl., Aneurysm of Aorta, Carc. of Larynx, Hypertension.
C 26	75	155	95	60	Art. scl., Chr. Bronch., Auric. Fibrillation, Myocard. Insuff., Chr. Arth.
27	78	128	75	53	Art. scl., Hypertrophied Prostate, Psychoneurosis.
28	76	130	55	75	Pernicious Anemia, Achylia Gastrica, Oral Sepsis.
C 29	77	105	70	35	Art. scl., Emphysema, Myocard, Degen, Auric Fibrillation.
C 30	79	110	60	50	Art. scl., Bronch. Pneumonia, Myocard. Insuff., Hypertrophied Prostate.
31	77	128	72	56	Gastric Ulcer, Secondary Anemia, Terminal Broncho-pneumonia.
C 32	79	156	92	64	Art. scl., Hypertension, Card. Hypertrophy, Senile Dementia.
33	76	164	75	89	Chron. Cholecystitis, Secondary Anem., Achylia Gastrica, Undernutrition.
R34	75	130	90	40	Diabetes Mellitus, Herpes Zoster, Oral Sepsis.
35	81	152	68	84	Hodgkin's Disease, Art. scl., Bilateral Hydrocele
C 36	75	130	80	50	Furunculosis, Carbuncles, Seborrheic Dermatitis, Mitr. and Myocard Insuff.
37	78	170	90	80	Art. scl., Emphysema, Carc. of Prostate.
R38	80	130	60	70	Hydrocele, Benign Hypertrophy of Prostate, Pyelitis
C 39	77	130	65	65	Art. scl., Myocard. Insuff., Auric. Fibrillation.
40	79	130	70	60	Art. scl., Hypertrophy of Prostate.
41	80	186	100	86	Epithelioma of Skin (Shoulder)
CR42	78	187	100	87	Angina Pectoris, Art. scl., Chr. Neph.
43	75	90	50	40	Art. scl., Senility, Bronch.-pneum., Emphysema.
C 44	76	146	72	74	Art. scl., Myocard. Insuff., Angina Pectoris, Oral Sepsis.
C 45	83	230	110	120	Senility, Art. scl., Hypertension, Myocard. Insuff., Cerebral Hemorrhage.
C 46	76	145	40	105	Syphilis of Aorta, Myocard. and Aortic Insuff., Transient Auric. Fibr.
CR47	78	140	78	62	Art. scl., Hypertension, Chronic Neph. and Myocard., Auric. Fibril.
48	76	118	70	48	Pleural Endothelioma
49	83	184	110	74	Art. scl., Hypertension, Hypertrophic Arthritis of Spine.
50	75	220	30	190	Art. scl., Hypertension, Cardiac and Prostatic Hypert., Hemipleg. Left.

NOTE.—The symbols C, R, and CR prefixed to the case numbers represent cardiac, renal, and cardio-renal respectively.

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A STUDY OF THE CORRELATION OF CERTAIN CHARACTERS OF THE BLOOD WITH BODY-WEIGHT, STATURE AND SURFACE-AREA

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Relatively great differences in respect to number of red cells, relative cell volume and amount of hemoglobin in the blood are commonly observed in healthy individuals. That these differences are real and are not to be explained on the basis of technical error is unquestioned. Sex, age, barometric pressure, and diurnal and temporary influences such as exercise, diet and vasomotor and psychic factors, have all been shown to produce an appreciable effect on the quantity of these constituents in the circulation at any given time. On the other hand, the relation of these inter-individual variations in the blood constituents to physical differences between the individuals concerned, has hitherto received little attention.

Are differences between individuals in respect to number of red cells, hemoglobin, and cell volume and differences between the same individuals in respect to external physical characteristics as represented by stature, body-weight, and surface-area, in any way correlated? Cabot (1) quoted Leichenstern as stating that lean, muscular people have on the whole more red blood cells per cubic millimeter than fat people, other things being equal. Recently Pearl and Miner (2) published a biometric study of the inter-individual variation in cell volume and age, body-weight and stature. Their material included 272 healthy males, 20 to 49 years of age. These writers found that individual differences in relative cell volume were not highly correlated with differences in either age, body-weight, or stature and that the regression of relative cell volume upon each of the other variables considered was small in amount and considerably less than the range of difference in cell volume compatible with physiological normality and health. The highest correlation, both gross and net, which relative cell volume exhibited was with stature.

The writer has recently reported red cell, hemoglobin, and cell volume determinations, as well as certain corpuscular measurements

in 100 healthy young men (3, 4) and 50 women (5, 5a). Although, statistically speaking, this series is small, since particular care was taken in the individual determinations, it has been thought worth while to analyze the material from a biometric standpoint.

The men and women examined were all students of Tulane University or Newcomb College who were in good health at the time of the examination and had no record of ill health in the past few years. They were selected only in this regard and in respect to age, the men examined ranging from 19 to 30 years, while the women were 17 to 30 years of age. In the present analysis age is considered as non-variant.

The technic and details of the blood determinations are given fully in the original papers and need not be repeated here. For each individual the following items were recorded or determined:

a. Age in years.

b. Body-weight in pounds.

c. Stature in inches.

d. Surface area in square meters. This was calculated from the body-weight and stature by means of the nomographic chart of Boothby and Sandiford (6).

e. Number of red blood corpuscles in millions per cubic millimeter.

f. Amount of hemoglobin in grams per 100 cubic centimeters of blood.

g. Cell volume, as cubic centimeters of packed red cells per 100 cubic centimeters of blood.

h. Average corpuscular volume in cubic microns.

i. Average corpuscular hemoglobin (amount of hemoglobin in the average red corpuscle) in micromicrograms ($\gamma\gamma$).*

j. Average corpuscular hemoglobin concentration (proportion of hemoglobin to volume of average red corpuscle), in per cent.

The letters $a, b, c, d, \ldots j$ will be used as subscript letters throughout this paper to denote the characters against which they stand in the above list.

The mean, median, standard deviation, and coefficient of variation of all the characters observed are summarized in Table I. Details of the distribution of individual variation in the characters studied have already been given in the earlier papers. From the data given in Table I, it is apparent that there is no significant skewness or asym-

* A micromicrogram is the millionth of a millionth part of a gram or gm. $\times 10^{-12}$.

TABLE 1

Variation Constants in 100 Normal Men and 50 Normal Women

CHARACTER	MEAN		MEDIAN		STANDARD DEVIATION		COEFFICIENT OF VARIATION %	
	Men	Women	Men	Women	Men	Women	Men	Women
a. Age in yrs.	23.8 ± .2	21.5 ± .3	23.4 ± .2	20.3 ± .4	2.4 ± .1	3.5 ± .2	10.2	16.30
b. Body-wt. in lb.	152 ± 1.3	125 ± 1.3	150 ± 1.7	125 ± 1.6	19.6 ± 1.2	13.0 ± .9	12.90	10.37
c. Stature in inches	69.2 ± .2	64.6 ± .2	69.6 ± .2	64.4 ± .3	2.7 ± .2	2.2 ± .2	3.95	3.45
d. Surface area in sq. m.	1.82 ± .01	1.60 ± .01	1.83 ± .01	1.60 ± .01	.13 ± .01	.08 ± .01	7.29	5.50
e. No. of red cells in millions per cu. mm.	5.854 ± .037	4.930 ± .027	5.770 ± .046	4.944 ± .034	.548 ± .026	.286 ± .019	9.36	5.80
f. Amt. of Hb.* in gm. per 100 cc.	15.90 ± .06	13.77 ± .09	15.97 ± .08	13.78 ± .12	.94 ± .05	.99 ± .07	5.92	7.20
g. Cell Vol. in cc. per 100 cc.	46.51 ± .26	39.50 ± .19	46.90 ± .33	39.27 ± .24	3.01 ± .19	2.03 ± .14	8.40	5.20
h. Corp. Vol. in μ^3	79.9 ± .5	79.1 ± .3	80.6 ± .6	80.7 ± .3	7.2 ± .3	3.9 ± .2	9.06	4.90
i. Corp. Hb.* $\gamma\gamma$	27.38 ± .17	27.96 ± .15	28.09 ± .21	28.00 ± .19	2.51 ± .12	1.55 ± .10	9.18	5.54
j. Corp. conc.* %	34.4 ± .2	35.2 ± .2	34.0 ± .3	35.1 ± .2	2.96 ± .14	1.81 ± .1	8.60	5.15

* The figures for hemoglobin, corpuscular hemoglobin and corpuscular concentration in men are based on the figures obtained before a new hemoglobinometer correction curve was determined (7). For the determination of correlation it was not considered worth while to add the necessary correction to each figure, as the error is a constant one.

metry in the variation of any of the characters, the median in each case being close to the mean. Pearl and Miner (2) have pointed out that in normal individuals variations in cell volume is relatively small in amount as compared with most other physiological characters of the organism. Judging by the values for coefficients of variation presented in Table I, this statement is likewise true for number of red blood cells, amount of hemoglobin, average corpuscular volume, corpuscular hemoglobin, and corpuscular concentration.

The correlation between the various characters considered has been determined by the method described by Pearl. Correlation between two variables may be measured by a coefficient which indicates the degree of correlation.

> It may take any value between 0, which is the result when there is no correlation at all between the variables, and either +1 or −1. When either of the latter values occurs, it means that the correlation is perfect, i.e. for every change in one of the variables there is a definite and constant proportional change in the value of the other. A positive correlation means that as one variable increases in value the other variable also increases and vice versa. A negative correlation means that as one variable increases the other decreases. (Pearl (8)).

It is essential that the correlation coefficient be considered in relation to its probable error. It is generally accepted among biometric workers that a constant which is smaller than twice its probable error is probably not significant. On the other hand, a constant which is three times its probable error is considered as probably significant, while one which is four or more times its error is certainly significant.

CORRELATION BETWEEN PHYSICAL CHARACTERS AND NUMBER OF RED CELLS, AMOUNT OF HEMOGLOBIN AND RELATIVE CELL VOLUME

The relation between variations in the external physical characters of the individuals observed and variations in number of red cells, amount of hemoglobin, and cell volume of the blood of these individuals is indicated by the zero order correlation coefficient given in Table II. It is evident that none of the correlation coefficients are high, but many of them are significantly greater than their probable errors. All the signs are positive, which indicates that the constituents of the blood here considered tend to increase with increasing body-weight, stature, and surface area. No single one of the gross blood constituents appears to be much more correlated with the several physical characters than another. For the men there appears to be a slightly greater degree of correlation between weight and the blood constituents

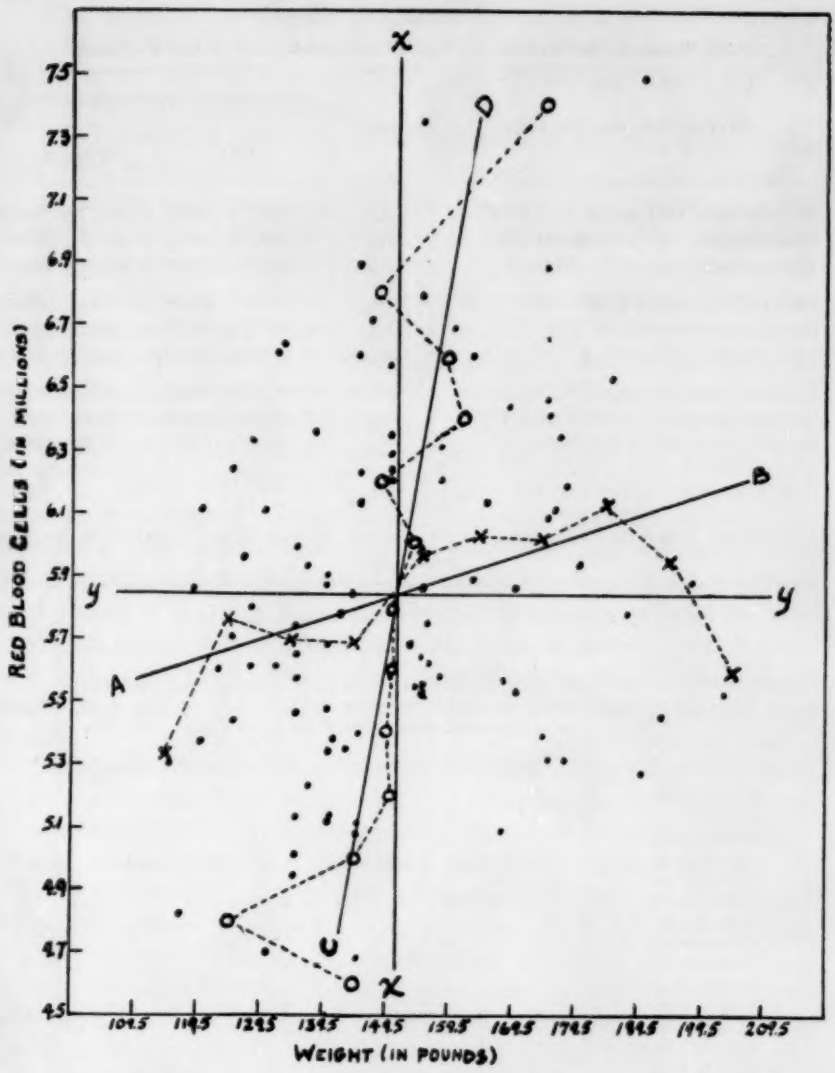

FIG. 1. SCATTER DIAGRAM SHOWING RELATION OF NUMBER OF RED BLOOD CELLS PER CU. MM. TO BODY-WEIGHT, IN 100 HEALTHY YOUNG MEN. The crosses are the means of the observed red blood cell arrays (observed regression of red blood cells on body-weight). *AB* is the calculated regression line of red blood cells on weight. The circles are the means of the observed weight arrays (observed regression of weight on red blood cells). *CD* is the calculated regression line of weight on red blood cells. *XX* gives the location on the body-weight scale of the mean of all 100 body-weights. *YY* gives the mean of all red cell counts on the red cell scale.

TABLE II

Coefficients of Correlation of Physical Characters and Other Variables

CHARACTERS CORRELATED	SYMBOL	COEFFICIENT OF CORRELATION	
		Men	Women
Body-weight and no. of R.B.C.'s	r_{be}	$+.2372 \pm .0637$	$+.2462 \pm .0896$
Body-weight and amount of Hb.	r_{bf}	$+.3042 \pm .0612$	$+.3144 \pm .0860$
Body-weight and cell volume	r_{bg}	$+.2455 \pm .0634$	$+.0644 \pm .0950$
Stature and no. of R.B.C.'s	r_{ce}	$+.1529 \pm .0659$	$+.1244 \pm .0939$
Stature and amount of Hb	r_{cf}	$+.0838 \pm .0670$	$+.2885 \pm .0875$
Stature and cell volume	r_{cg}	$+.2600 \pm .0622$	$+.2465 \pm .0896$
Surface area and no. of R.B.C.'s	r_{de}	$+.1715 \pm .0662$	$+.2959 \pm .0870$
Surface area and amount of HB	r_{df}	$+.2309 \pm .0639$	$+.3866 \pm .0811$
Surface area and cell volume	r_{dg}	$+.2890 \pm .0618$	$+.1628 \pm .0929$

TABLE III

Correlation Table for Body-Weight and Number of Red Blood Cells. 100 *Healthy Young Men*

RED BLOOD CELLS MILLIONS PER CU. MM.	BODY-WEIGHT (POUNDS)										
	109.5 to 119.4	119.5 to 129.4	129.5 to 139.4	139.5 to 149.4	149.5 to 159.4	159.5 to 169.4	169.5 to 179.4	179.5 to 189.4	189.5 to 199.4	199.5 to 209.4	Totals
4.50 to 4.69	..	..	..	1	..	..	..	..	..	..	1
4.70 to 4.89	1	..	1	..	..	..	..	..	..	..	2
4.90 to 5.09	..	..	2	1	..	1	..	..	..	..	4
5.10 to 5.29	..	..	2	3	1	..	..	..	1	..	7
5.30 to 5.49	1	2	1	5	..	..	3	..	1	..	13
5.50 to 5.69	..	3	3	1	9	1	1	..	..	1	19
5.70 to 5.89	1	2	1	3	2	1	1	1	1	..	13
5.90 to 6.09	..	1	2	1	2	..	2	1	..	..	9
6.10 to 6.29	..	2	1	2	4	1	1	..	..	..	11
6.30 to 6.49	..	..	1	..	2	1	3	..	..	..	7
6.50 to 6.69	..	..	2	1	1	2	2	1	..	..	9
6.70 to 6.89	..	..	..	2	1	..	..	..	..	..	3
6.90 to 7.09	..	..	..	..	..	..	..	..	..	..	0
7.10 to 7.29	..	..	..	..	..	..	..	..	..	..	0
7.30 to 7.49	..	..	..	..	1	..	..	..	1	..	2
Totals	3	10	16	20	23	7	13	3	4	1	100

recorded than there is between these constituents and stature or surface area, while among the women the correlation appears to be greatest with surface area.

The degree of correlation in a typical example is indicated graphically by the scatter diagram shown in Figure 1, as well as by Table III, which is the table of correlation for body-weight and number of red cells in the 100 men examined.

The correlation coefficient is only of value as a measure of correlation when the regression of one variable on another is linear. Linearity of regression is measured by the formula $\zeta = \eta^2 - r^2$ where η is the correlation ratio, and r the correlation coefficient. The correlation ratio is a constant proposed by Pearson (9) for the measurement of the degree of correlation existing in a particular case regardless of whether the regression is linear or not.

Correlation ratios for body-weight, stature, and surface area, on the one hand, and number of red cells, amount of hemoglobin, and cell volume on the other hand are given in Table IV. In this table are found crude η as well as η corrected for the number of arrays, and $\sqrt{(k-1)/N} \pm 0.67449/\sqrt{N}$, which is the mean value of η in samples from an uncorrelated population, together with its probable error. A number of these correlation ratios are lower than the correlation ratio which would be found in a totally uncorrelated population. Of those that exceed $\sqrt{(k-1)/N}$ the difference is in no case as great as three times the probable error of η for samples from an uncorrelated population. In no case is $\zeta = \eta^2 - r^2$ significantly greater than its probable error, from which it may be concluded that regression is in each case linear.

The degree of correlation between the external physical characters of the individuals here considered and the gross constituents of the blood may be indicated by considering the reduction in the variability of one character when a correlated variable is held constant. In Table V the variability of the several characters considered is indicated by their standard deviation. The reduction in variability resulting when a related character is held constant has been measured by a technic described by Pearl (10) and is indicated by the "reduced standard deviation." The actual reduction in variability, measured in per cent, is given in the last two columns of the table. Thus, the standard deviation of the number of red cells among the men is 0.548 million. The correlation of number of red cells and weight in the men examined is such that, were all the men examined of the same weight, the standard deviation would become 0.532 million, a reduction of 2.9 per cent. It is clearly evident from the figures shown in Table V that, in the samples here examined, the associational tendencies of

TABLE IV

Correlation Ratios for Physical Characters and Other Variables

CHARACTERS CORRELATED	SYMBOL	MEN				WOMEN			
		Crude η	Corrected η	$\sqrt{(k-1)/N} \pm 0.67449/\sqrt{N}$	$\zeta = \eta^2 - r^2$	Crude η	Corrected η	$\sqrt{(k-1)/N} \pm 0.67449/\sqrt{N}$	$\zeta = \eta^2 - r^2$
Body-wt. and R.B.C.	η_{be}	.3329	Imaginary	.3742 ± .0674	.0545 ± .0299	.3601	.2723	.3162 ± .0954	.0691 ± .0484
Body-wt. and Hb	η_{bf}	.4266	.3743	.3000 ± .0674	.1078 ± .0418	.5423	.4448	.4000 ± .0954	.1953 ± .0756
Body-wt. and cell vol.	η_{bg}	.3341	.1520	.3316 ± .0674	.0503 ± .0288	.2609	.1711	.2828 ± .0954	.0640 ± .0452
Stature and R.B.C.	η_{ce}	.3115	Imaginary	.3742 ± .0674	.0737 ± .0340	.2895	.0640	.3464 ± .0954	.0638 ± .0481
Stature and Hb	η_{cf}	.3390	.1948	.3162 ± .0674	.1079 ± .0395	.5448	.4482	.4000 ± .0954	.2136 ± .0782
Stature and Cell vol.	η_{cg}	.3545	.1981	.3317 ± .0674	.0481 ± .0283	.2583	.1668	.2828 ± .0954	.0059 ± .0146
Surface area and R.B.C.	η_{de}	.2926	.1364	.3742 ± .0674	.0562 ± .0770	.3638	.2378	.3464 ± .0954	.0448 ± .0387
Surface area and Hb	η_{df}	.3035	.1147	.3162 ± .0674	.0388 ± .0256	.5999	.5640	.4000 ± .0954	.2104 ± .0725
Surface area and cell vol.	η_{dg}	.4099	.2927	.3316 ± .0674	.0845 ± .0420	.2472	.1483	.2828 ± .0954	.0346 ± .0343

external physical characteristics and the gross blood constituents were very small indeed.

TABLE V

Reduction in Variability when Related Variable Is Held Constant

VARIABLE CONSIDERED	VARIABLE HELD CONSTANT	VARIABILITY (EXPRESSED BY STANDARD DEVIATION)		REDUCED STANDARD DEVIATION		VARIABILITY REDUCED BY	
		Men	Women	Men	Women	Men %	Women %
No. of R.B.C.	Weight	0.548 million R.B.C.	0.286 million R.B.C.	0.532 million R.B.C.	0.272 million R.B.C.	2.9	3.1
No. of R.B.C.	Stature	0.548 million R.B.C.	0.286 million R.B.C.	0.542 million R.B.C.	0.284 million R.B.C.	1.2	0.8
No. of R.B.C.	Surface area	0.548 million R.B.C.	0.286 million R.B.C.	0.540 million R.B.C.	0.273 million R.B.C.	1.5	4.5
Amt. of Hb.	Weight	0.94 gm. Hb.	0.99 gm. Hb.	0.895 gm. Hb.	0.94 gm. Hb.	4.7	5.1
Amt. of Hb.	Stature	0.94 gm. Hb.	0.99 gm. Hb.	0.937 gm. Hb.	0.95 gm. Hb.	0.3	4.2
Amt. of Hb.	Surface area	0.94 gm. Hb.	0.99 gm. Hb.	0.915 gm. Hb.	0.91 gm. Hb.	2.7	7.8
Cell volume	Weight	3.91 cc.	2.03 cc.	3.78 cc.	2.03 cc.	3.1	0.2
Cell volume	Stature	3.91 cc.	2.03 cc.	3.77 cc.	1.98 cc.	3.3	3.1
Cell volume	Surface area	3.91 cc.	2.03 cc.	3.74 cc.	2.00 cc.	4.3	1.3

A more concrete conception of the degree of correlation present can be gained from information derived from a consideration of regression equations. The equations for the regression of the various blood characters upon the external physical characters in the men examined are given in absolute terms in Table VI (a bar over a letter denoting

TABLE VI

Regression Equations for 100 *Healthy Young Men*

REGRESSION OF	REGRESSION EQUATION
R.B.C.'s on weight	$\bar{e} = 4.84 + .0066b$
Hb. on weight	$\bar{f} = 13.67 + .0147b$
Cell volume on weight	$\bar{g} = 40.52 + .0388b$
R.B.C.'s on stature	$\bar{e} = 3.72 + .0308c$
Hb. on stature	$\bar{f} = 14.21 + .0244c$
Cell volume on stature	$\bar{g} = 21.10 + .3668c$
R.B.C.'s on surface area	$\bar{e} = 4.54 + .711d$
Hb. on surface area	$\bar{f} = 12.96 + 1.61d$
Cell volume on surface area	$\bar{g} = 31.16 + 8.41d$

a mean value). For these equations the following deductions can be made:

1. A difference in increasing body-weight of 88 pounds between the limiting weights in the sample of 115 and 203 pounds has associated with it an increase in number of red cells of 0.58 million per cu. mm., 1.29 gm. of hemoglobin and 3.42 cc. of packed red cells per 100 cc. of blood.

2. A difference in increasing stature of 12 inches, between the limiting heights of 62 and 74 inches, has associated with it an increase in number of red cells of 0.37 million per cu. mm., 0.30 gm. of hemoglobin and 4.40 cc. of packed red cells per 100 cc. of blood.

3. A difference in increasing surface area of 0.64 sq. m., between the limiting surface areas of 1.51 and 2.15 sq. m. in the sample, has associated with it an increase in number of red cells of 0.46 million per cu. mm., 1.03 gm. of hemoglobin and 5.38 cc. of packed red cells per 100 cc. of blood.

Summing up what has already been presented, it may be said that, for the sample here considered, there appears, on the basis of correlation coefficients, to be a very slight degree of correlation between the physical characters of an individual as represented by body-weight, stature, and surface area, and the gross constituents of his blood as represented by number of red cells, amount of hemoglobin, and relative cell volume. The regression of these variables on one another is linear. The degree of correlation is, however, quite small.

Other zero-order correlation coefficients necessary for the computation of net correlation are given in Table VII. The following net or

TABLE VII

Zero-Order Correlation Coefficients

CHARACTERS CORRELATED	SYMBOL	COEFFICIENTS OF CORRELATION	
		Men	Women
Stature and body-weight	r_{cb}	$+.5262 \pm .0490$	$+.5496 \pm .0666$
Amount of Hb. and cell volume	r_{fg}	$+.3653 \pm .0585$	$+.5925 \pm .0619$
No. of red cells and hemoglobin	r_{cf}	$+.3547 \pm .0742$	$+.6732 \pm .0758$
No. of red cells and cell volume	r_{cg}	$+.5100 \pm .0499$	$+.6559 \pm .0544$

partial correlation coefficients have been derived from the data presented:

Net correlation between body-weight and number of red cells, with stature, hemoglobin and cell volume constant, $r_{bc.sfv} = +.0928 \pm .0669$

Net correlation between body-weight and cell velume with stature, number of red cells, and hemoglobin constant, $r_{bv.scf} = -.0468 \pm .0673$

Neither of these coefficients is significant. It is to be expected that, when the effects of other variables are eliminated, the degree of correlation is diminished, since the number of red blood cells, amount of hemoglobin, and cell volume tend to act as a physiological unit rather than separately.

CORRELATION BETWEEN PHYSICAL CHARACTERS AND ERYTHROCYTIC MEASUREMENTS

By a technic which has been described elsewhere (4), it is possible from a knowledge of the number of red cells, the amount of hemoglobin, and the cell volume of a given sample of blood, to calculate the volume of the average red corpuscle, the amount, by weight, of hemoglobin it contains, and the proportion of its substance taken up by the hemoglobin. These values are termed, respectively, corpuscular volume, corpuscular hemoglobin and corpuscular concentration. They have been determined from the data reported for the 100 men and 50 women examined. Details of these determinations have already been pub-

TABLE VIII

Coefficients of Correlation between Physical Characters and Erythrocytic Measurements

CHARACTERS CORRELATED	SYMBOL	COEFFICIENT OF CORRELATION	
		Men	Women
Body-weight and corpuscular volume	r_{bh}	$+.0210 \pm .0670$	$-.1868 \pm .0921$
Body-weight and corpuscular Hb	r_{bi}	$+.1015 \pm .0668$	$+.0538 \pm .0951$
Body-weight and corpuscular concentration	r_{bj}	$-.0701 \pm .0671$	$+.3281 \pm .0851$
Stature and corpuscular volume	r_{ch}	$+.0664 \pm .0672$	$-.0349 \pm .0953$
Stature and corpuscular Hb	r_{ci}	$-.1017 \pm .0668$	$+.0369 \pm .0953$
Stature and corpuscular concentration	r_{cj}	$-.2187 \pm .0642$	$+.0940 \pm .0945$
Surface area and corpuscular volume	r_{dh}	$+.0656 \pm .0672$	$-.0806 \pm .0948$
Surface area and corpuscular Hb	r_{di}	$-.0548 \pm .0673$	$+.1625 \pm .0702$
Surface area and corpuscular concentration	r_{dj}	$-.1374 \pm .0662$	$+.2297 \pm .0904$

TABLE IX

Correlation Ratios for Physical Characters and Erythrocytic Measurements

CHARACTERS CORRELATED	SYMBOL	MEN				WOMEN			
		Crude η	Corrected η	$\sqrt{(k-1)/N} \pm 0.67449/\sqrt{N}$	$\zeta - \eta^2 - r^2$	Crude η	Corrected η	$\sqrt{(k-1)/N} \pm 0.67449/\sqrt{N}$	$\zeta = \eta^2 - r^2$
Body-wt. and corp. vol	η_{bk}	.2545	Imaginary	.3000 ± .0674	.0644 ± .0320	.3532	.2970	.2828 ± .0954	.0898 ± .0547
Body-wt. and corp. Hb	η_{bi}	.2422	Imaginary	.3000 ± .0674	.0484 ± .0282	.1751	Imaginary	.3162 ± .0954	.0018 ± .0798
Body-wt. and corp. conc	η_{bj}	.2792	.0927	.3000 ± .0674	.0731 ± .0338	.4108	.3407	.3162 ± .0954	.0611 ± .0457
Stature and Corp. vol	η_{ok}	.2842	.1078	.3000 ± .0674	.0764 ± .0344	.2817	.2026	.2828 ± .0954	.0782 ± .0512
Stature and Corp. Hb	η_{oi}	.4497	.3056	.3742 ± .0674	.1918 ± .0531	.3102	Imaginary	.4000 ± .0954	.0948 ± .0559
Stature and Corp. conc	η_{oj}	.3669	.0728	.3873 ± .0674	.0868 ± .0380	.4002	.2135	.4000 ± .0954	.1513 ± .0653
Surface area and corp. vol	η_{dk}	.2461	Imaginary	.3000 ± .0674	.0563 ± .0311	.2467	.1476	.2828 ± .0954	.0544 ± .0421
Surface area and corp. Hb	η_{di}	.4259	.2646	.3742 ± .0674	.1784 ± .0468	.4251	.2620	.4000 ± .0954	.1543 ± .0637
Surface area and corp. conc	η_{dj}	.3769	.1179	.3873 ± .0674	.1232 ± .0416	.4384	.2864	.4000 ± .0954	.1394 ± .0619

TABLE X

Reduction in Variability when Related Variable is Held Constant

VARIABLE CONSIDERED	VARIABLE HELD CONSTANT	VARIABILITY (EXPRESSED BY STANDARD DEVIATION)		REDUCED STANDARD DEVIATION		VARIABILITY REDUCED BY	
		Men	Women	Men	Women	Men %	Women %
Corp. vol.	Weight	7.2 cu. μ	3.9 cu. μ	7.199 cu. μ	3.83 cu. μ	0	1.8
Corp. vol.	Stature	7.2 cu. μ	3.9 cu. μ	7.18 cu. μ	3.90 cu. μ	0.2	0.1
Corp. vol.	Surface area	7.2 cu. μ	3.9 cu. μ	7.18 cu. μ	3.89 cu. μ	0.2	0.3
Corp. Hb.	Weight	2.51 $\gamma\gamma$	1.55 $\gamma\gamma$	2.50 $\gamma\gamma$	1.55 $\gamma\gamma$	0.5	0.1
Corp. Hb.	Stature	2.51 $\gamma\gamma$	1.55 $\gamma\gamma$	2.50 $\gamma\gamma$	1.55 $\gamma\gamma$	0.5	0.1
Corp. Hb.	Surface area	2.51 $\gamma\gamma$	1.55 $\gamma\gamma$	2.50 $\gamma\gamma$	1.53 $\gamma\gamma$	0.1	1.3
Corp. conc.	Weight	2.96%	1.81%	2.95%	1.71%	0.2	5.5
Corp. conc.	Stature	2.96%	1.81%	2.89%	1.80%	2.4	0.4
Corp. conc.	Surface area	2.96%	1.81%	2.93%	1.76%	0.9	2.7

lished (4, 5*a*). The mean, median, standard deviation, and coefficient of variation of these values have been given in Table I.

In Table VIII are found the zero-order coefficients of correlation between body-weight, stature and surface area and these erythrocytic measurements. Examination of these coefficients reveals the fact that, considering probable errors, there is absolutely no correlation between external physical characters and corpuscular volume or corpuscular hemoglobin. Although the coefficient of correlation between corpuscular concentration and stature in men, and body-weight in women, is more than three times its probable error, since such a correlation is not consistently observed, it is probably insignificant.

Corresponding correlation ratios are given in Table IX. None of these values can be considered significant having regard to probable errors. Examining linearity of regression, we find that in only two instances is $\zeta = \eta^2 - r^2$ significantly greater than its probable error. It should be remembered, however, that this value does not take account of the effect of number of arrays on the value of η.

The reduction in the variability of the erythrocytic measurements when each of the external physical characters is, in turn, held constant, has been calculated and is shown in Table X. It is apparent that, except in a few instances, the reduction in variability is inappreciable. Again, to cite a concrete example, from the regression equations listed in Table XI, it appears that a difference in increasing body-weight of 88 pounds, between the limiting weights for the men of the sample

TABLE XI

Regression Equations. 100 *Healthy Young Men*

REGRESSION OF	REGRESSION EQUATION
Corpuscular volume on body-weight	$\overline{h} = 78.54 + .0079b$
Corpuscular Hb. on body-weight	$\overline{i} = 25.41 + .0131b$
Corpuscular concentration on body-weight	$\overline{j} = 35.98 - .0105b$

of 115 and 203 pounds, has associated with it an increase of 0.7 cu. μ in corpuscular volume and 1.15$\gamma\gamma$ in corpuscular hemoglobin, while there is a decrease of 0.9 per cent in corpuscular concentration.

It is interesting to compare the coefficients expressing the correlation between the gross blood constituents (number of red blood cells, amount of hemoglobin, and cell volume) and the external physical characters here considered (Table II), and those which denote the relation of the erythrocytic measurements (average corpuscular volume, corpuscular hemoglobin and corpuscular concentration) with these physical characters (Table VIII). While the latter coefficients are small and statistically not significant, the former are of much greater magnitude and significantly greater than their probable errors. It appears then, that number of red cells, amount of hemoglobin, and relative cell volume are to a small degree related to and variant with the biological reference-frame represented by body-weight, stature, and surface area, whereas average corpuscular volume, corpuscular hemoglobin, and corpuscular concentration are in this respect entirely invariant. This fact is of special interest in view of the marked variation in corpuscular volume and hemoglobin which is seen in various types of anemia.

SUMMARY

This paper presents the results of a study of the correlation between certain erythrocytic components of the blood,—namely, number of red blood cells, amount of hemoglobin, relative cell volume, average corpuscular volume, average corpuscular hemoglobin, and average corpuscular concentration,—and body-weight, stature and surface-area in 100 normal men 19 to 30 years of age and 50 women 17 to 30 years of age. As a result of this study it appears that:

1. The distribution of variation about their mean values of all the characters of the blood above mentioned is substantially symmetrical and relatively small in amount.

2. Individual differences in number of red cells, amount of hemoglobin, and cell volume are, in gross, slightly correlated with individual differences in body-weight, stature, and surface-area. The sign of correlation is positive. The regression of these variables upon each other is linear and quite small in amount as compared with the range of difference in these characters observed in normal individuals. Within the range of the sample, number of red cells, amount of hemoglobin, and relative cell volume were, for the men, most highly correlated with weight and, for the women, with surface-area.

3. Individual differences in average corpuscular volume, average corpuscular hemoglobin, and average corpuscular concentration are not at all associated with differences in body-weight, stature, and surface-area. This fact is of special interest in view of the marked variation in corpuscular volume and hemoglobin associated with various types of anemia.

To Dr. Raymond Pearl the writer wishes to express gratitude for his kindly interest and advice, and indebtedness for his book on medical biometry and statistics, which has proved invaluable in this study. The writer also wishes to thank Dr. J. R. Miner for his valuable criticism.

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In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Raymond Pearl, Institute for Biological Research, 1901 East Madison Street, Baltimore, Maryland, U. S. A.

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